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## HEMORRHAGE INTO THE ESOPHAGUS AT BIRTH AND IN THE ADULT.

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It has been shown that periesophageal infection can result in narrowing of the terminal portion of the esophagus by an increase of fibrous tissue around the esophagus or in the esophagus itself. Such fibrosis has been found in the terminal portion of the esophagus and in its middle third. Excepting cervical exostoses which produce webs of the esophagus, no cause has been found for a common type of stricture found below the cricoid cartilage, which seems on examination to be due to a periesophageal fibrosis rather than to a web or actual stricture in the lumen of the esophagus itself.

*The Title of the Paper:* The paper should have a double title. This, however, makes an awkward combination so the second part of the title, "Infection of the Gall Bladder as a Cause of Cardiospasm," was omitted and is given here.

*The Purpose of the Paper:* The purpose of the paper is to give a cause of periesophageal fibrosis; to show that the esophagus can be infected before birth; that peri- and intraesophageal hemorrhages occur before birth, associated with toxemia or sepsis; that hemorrhages into the esophagus and into its sheath occur in hemorrhagic

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disease of the newborn; that infection of the gall bladder and atrophic cirrhosis of the liver are associated with pathological changes in the terminal portion of the esophagus.

*Material of the Paper:* The material of the paper consists of specimens of the esophagus from 20 babies dead at birth and two infants; also of 43 specimens of the terminal portion of the adult esophagus.

I am greatly indebted to Dr. Charles F. Branch, of the Massachusetts Memorial Hospital, of Boston, for his very great kindness in sending me practically all the specimens upon which the paper is based. For the remainder of the specimens I am indebted to Dr. William Freeman, Beverly Hospital, Beverly Mass.; Dr. William Moran, Salem Hospital, Salem, Mass.; and Dr. T. B. Mallory, Massachusetts General Hospital.

The records of the cases were also furnished by Dr. Branch.

Dr. William E. Murphy, House Officer in the Otolaryngological Service, Massachusetts Eye and Ear Infirmary, took great pains to collect the specimens and get them to me.

A 30 24. Seven months fetus.
A 30 25. Infant 14 months. Lymphatic leukemia.
A 30 27. Status lymphaticus.
A 30 115. Intracranial hem. and adrenal hem. Age 23 hours.
A 30 134. Intracranial hem. and adrenal hem.
A 30 137. Premature. Baby lived 12 hours.
A 30 139. Pyloric stenosis. Marasmus. Rhamstead operation.
A 30 143. Congenital syphilis and sepsis. Baby 21 days old.
A 30 144. Still birth. Strangulated cord.
A 30 146. Intracranial hemorrhage. Age 2 days.
A 30 152. Premature. 7½ months. Age 7 hours. Question of hemorrhagic disease of newborn.
A 30 153. Pyloric stenosis. Marasmus.
A 30 155. Premature. 8 months. Age 2 days. Mother T. B. Toxemia of pregnancy. Induced labor.
A 30 162. Atelectasis.
A 30 171. Infant. Bronchopneumonia.
A 30 172. Premature.
A 31 4. Seven months fetus.
A 31 9. Atelectasis.
A 31 21. Petechial hemorrhages heart, lungs and brain.
A 31 26. Hemorrhagic disease of the newborn. Age 28 hours.
No number. Four months fetus. Dr. Eades.
No number. Term baby. Mother died of eclampsia. Dr. Eades.

Premature, 6
Term, 14
Infants, 2

22

From these 22 cases the following grouping can be made:

| | *No. Cases* |
|---|---|
| Intracranial hemorrhage | 1 |
| Intracranial hemorrhage and adrenal hemorrhage | 2 |
| Hemorrhagic disease of the newborn | 3 |
| Dead babies from eclamptic mothers | 2 |
| Pyloric stenosis | 2 |
| Congenital syphilis | 1 |
| Status lymphaticus | 1 |

The above list, it will be seen, includes six hemorrhagic cases.

Forty-three specimens of the terminal portion of the esophagus were examined.

In the following cases the history in detail is given only in a few of the more important ones.

*The Order of the Paper:* The subjects discussed and the order in which they are presented are the same as in the paragraph stating the purpose of the paper with the exception that the first subject there mentioned is taken up last.

PATHOLOGICAL CHANGES BEFORE TERM.

A 30 172. Premature. *Microscopical Findings:* Marked periesophageal hemorrhage. Small round-cell infiltration. No change in Auerbach's plexus.

A 31 9. Premature; seven months. Atelectasis. *Microscopical Findings:* Periesophageal hemorrhage. No change in Auerbach's plexus.

A 30 137. Premature. Baby lived 12 hours. *Microscopical Findings:* Greatly dilated capillaries of the esophagus. Hemorrhage into nerve vagus or phrenic. No change in Auerbach's plexus (see Figs. 1, 2, 3 and 4).

A 30 155. Premature; eight months. Age 2 days. Male, white. *Pathological Diagnosis:* Partial patent ductus arteriosus. Maternal active tuberculosis. Maternal toxemia of pregnancy. *History:* Mother: Mother came to the hospital with toxemia of pregnancy, vomiting, vertigo, headache. Had been vomiting since beginning of pregnancy. Blood pressure, 136 over 84. Blood chemistry, 15.3 m.g. of uric acid. Leukocytes, 18,800; 88 per cent polymorphonuclear leukocytes. Active pulmonary tuberculosis on physical examination. X-ray

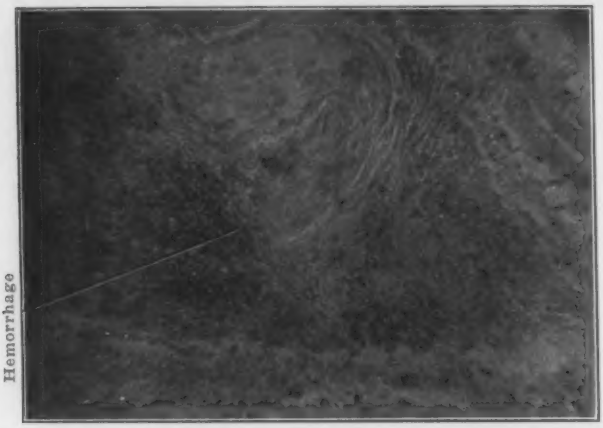



Fig. 1. A-30-137. Premature. Baby lived 12 hours. Section taken below cardia. Hemorrhage into nerve (vagus or phrenic), no hemorrhage into Auerbach's plexus.

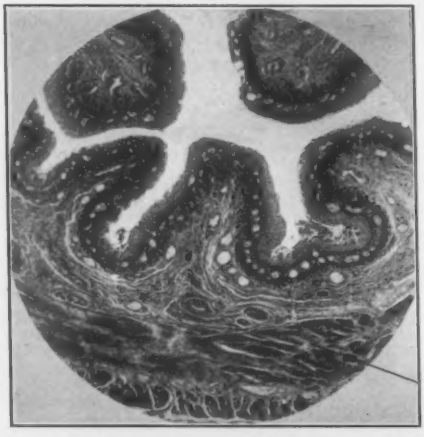



Fig. 2. A-30-137. Premature. Section taken at the cardia. It shows enlarged capillaries, and beginning fibrosis of the circular muscular layer.

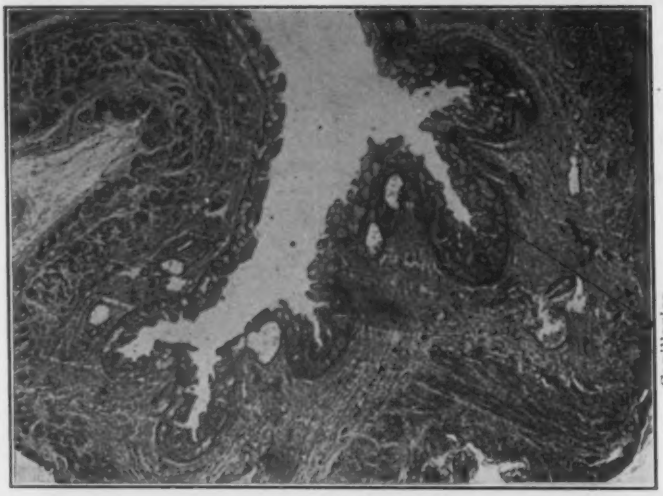



Fig. 3. A-30-137. Premature. Section taken below the cardia. It shows dilated capillaries.

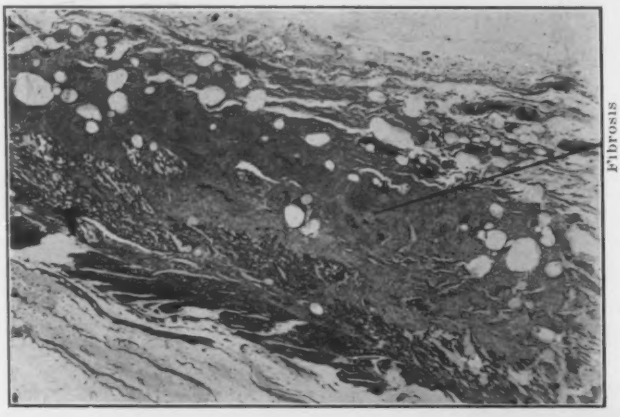



Fig. 4. A-30-137. Premature. Section taken below the cardia. It shows no epithelium. There is great dilatation of the capillaries and a great increase of the subepithelial connective tissue. There is disorganization of the circular muscular layer.

Fig. 5. A-30-155. Premature, 8 months. Age 2 days. Mother had active tuberculosis of the lungs and toxemia of pregnancy. Section of esophagus above the cardia. It shows great dilatation of all the vessels. There is hemorrhage into the crus, and periesophageal hemorrhage. In the esophagus there is subepithelial hemorrhage and much hemorrhage into the muscles. There is hemorrhage about, but not into Auerbach's plexus.

Fig. 6. A-30-155. Premature, 8 months. High power. The mother had tuberculosis of the lungs and toxemia of pregnancy. See Fig. 5.

positive. Sputum negative. Delivery: manual dilatation. Mid. forceps. Easy delivery.

Baby: Baby weighed 3 pounds, 15½ ounces. Put on artificial feeding immediately. On second day baby suddenly became cyanotic and died. Physical examination and clinical progress up to the time of death, negative.

*Microscopical Findings in the Esophagus of Baby:* The esophagus is riddled with dilated vessels, mostly veins. There are many large intramuscular hemorrhages. There is abundant periesophageal hemorrhage. There is an increase of connective tissue in and about the esophagus (see Figs. 5, 6 and 7).

Fig. 7. A-30-155. Premature. Section taken at the cardia. Mother had active tuberculosis and toxemia of pregnancy. Hemorrhage into epithelium. Periesophageal vessels filled with degenerating polymorphonuclear leukocytes.

PATHOLOGICAL CHANGES AT TERM — HEMORRHAGIC DISEASE OF THE NEWBORN.

A 30 152. Question of hemorrhagic disease of the newborn. *Microscopical Findings:* Marked periesophageal hemorrhage. No change in Auerbach's plexus (see Fig. 8).

A 31 21. Petechial hemorrhages of brain, heart and lungs. Edema of brain. *Microscopical Findings:* Marked periesophageal hemorrhage. Slight intramuscular hemorrhage. No change in Auerbach's plexus.

A 31 26. Twenty-eight hours old. Male, white. *Pathological Diagnosis:* Petechial hemorrhages, both lungs. *History:* Mother: Multipara. Two children. Past history not noteworthy. Past pregnancies normal. Physical examination negative. Delivery easy, high

forceps. Baby: Baby well until day after birth, when it became suddenly cyanotic while feeding. Tracheal catheter brought up blood. Carbon dioxid given, but baby died without recovering from cyanosis. *Microscopical Findings:* Periesophageal hemorrhage. No change in Auerbach's plexus.

INTRACRANIAL HEMORRHAGE AND HEMORRHAGE INTO THE ESOPHAGUS.

A 30 115. Age 23 hours. Male, white. *Pathological Diagnosis:* Intracranial hemorrhage. Partial atelectasis. Hypertrophied thymus. Edema. Left adrenal hemorrhage. *History:* Mother: Multipara. Ten children. Past history not noteworthy. Past pregnancies

Fig. 8. A-30-152. Hemorrhage into the epithelium of stomach. It would take but little trauma, chemical or physical, to cause gastric hemorrhage from an epithelium infiltrated with blood to the extent shown in the illustration.

normal. Physical examination negative. Baby: Normal delivery. Respiration established with difficulty. Thirty c.c. of maternal blood given. X-ray of chest showed enlarged thymus. First X-ray dose given to thymus. Baby died a respiratory death (see Fig. 9). *Microscopical Findings:* Subepithelial hemorrhage. Light periesophageal hemorrhage. Perigastric hemorrhage. Increase of fibrosis in circular layer of esophagus. Longitudinal layer early obliterated by fibrosis. Hemorrhage into Auerbach's plexus.

A 30 134. Intracranial and adrenal hemorrhage. *Microscopical Findings:* Periesophageal hemorrhage.

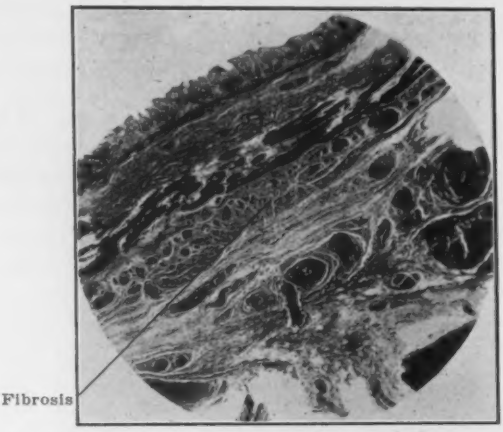
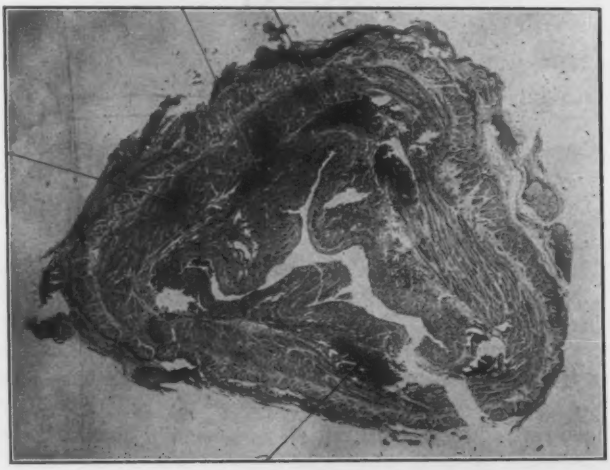



Fig. 9. A-30-115. Cardia. X15. Age 23 hours. Intracranial hemorrhage and hemorrhage into the esophagus. Microscopical Findings: Subepithelial hemorrhage. Light periesophageal hemorrhage. Perigastric hemorrhage. Increase of fibrosis of circular layer. Longitudinal layer nearly obliterated by fibrosis. Hemorrhage into Auerbach's plexus. Section shows increased fibrosis of the circular layer and partial obliteration of the longitudinal layer by fibrosis. This case shows, therefore, fibrosis of the esophagus at birth.



All markers point to hemorrhage.

Fig. 10. A-30-146. Baby lived 36 hours. Intracranial hemorrhage. Hemorrhage into esophagus (subepithelial and intramuscular). Periesophageal hemorrhage (abundant). Large intramuscular hemorrhages disrupting muscular fibres. Question of hemorrhage into Auerbach's plexus. Question of subepithelial infiltration. Hemorrhage into the muscles of the stomach.

A 30 146. Age 2 days. Male, white. *Pathological Diagnosis:* Intracranial hemorrhage. Partial atelectasis. Primary bronchopneumonia. *History:* Mother: Multipara. Seven children. Past history not noteworthy. Past pregnancies normal. Present pregnancy normal. Delivery normal. Baby: Baby developed symptoms of intracranial pressure. Temperature, 107.8°, with convulsions lasting seven minutes. Spinal tap done easily. Clear fluid obtained. Convulsions stopped. Baby died a respiratory death. *Microscopical Findings in Esophagus of Baby:* Very large intramuscular hemorrhages. In places the esophagus is nearly torn across by the hemorrhage (see Fig. 10).

Fig. 11. A-30-143. Age 21 days. Low power. Congenital syphilis and sepsis. Dilatation of vessels. Intramuscular hemorrhage. See Fig. 12.

A 30 143. Age 21 days. *Postmortem Diagnosis:* Congenital syphilis. Acute ulcerative pharyngitis and tonsillitis. Pneumonia. Central necrosis of liver. Marasmus. *Clinical History:* Mother had positive Wassermann, Kahn and Hinton tests at time of delivery. Mother otherwise normal. Delivery normal. Baby developed macular-pustular rash all over body. Liver slightly enlarged. Physical examination otherwise negative. Wassermann, Kahn and Hinton tests strongly positive. Antisyphilitic treatment given. X-ray of chest negative. Developed pneumonia on thirteenth day. Died on twenty-first day.

*Microscopical Findings:* Esophagus: Marked dilatation and filling of vessels. Intramuscular hemorrhage. Stomach: The lower

third of the stomach about the greater curvature stood out at a thickened patch at least three times thicker than the fundus. There were almost no rugae at the fundus. They were numerous, however, over the thickened area. Microscopical examination of the specimen from the fundus showed a submucosal infiltration with small round cells and a slight caseation. There was a thin hemorrhage throughout the whole mucosa. The perigastric vessels were choked with small lymphocytes and polymorphonuclear leukocytes. In one place the mucosa of the stomach was replaced by a patch of stratified squamous epithelium.

The microscopical examination of the thickened area showed an extensive change of the mucosa into squamous epithelium. There

Fig. 12. A-30-143. Congenital syphilis and sepsis. Age 21 days. High power. The section shows dilatation of all the vessels. The vessels are filled with polymorphonuclear leukocytes. There is intramuscular hemorrhage.

were many glands adjacent to this area. These showed much hemorrhage. The perigastric vessels about the thickened area were also filled with small lymphocytes and polymorphonuclear leukocytes (see Figs. 10 and 16).

Summing up the foregoing cases, it will be seen that in the premature group of four, two had periesophageal hemorrhage, one hemorrhage into a nerve, and one, the case of toxemia of pregnancy (A 30 155), very extensive hemorrhages all through the esophagus.

I examined one other baby from a mother who died of eclampsia. The esophagus of this baby, however, showed no pathological changes.

In the next group the babies were dead at term. In the first three the diagnosis of hemorrhagic disease of the newborn was made. In all periesophageal hemorrhage was found.

In the second three there was intracranial hemorrhage. All had periesophageal hemorrhage, and one (A 30 146) had multiple large hemorrhages into the musculature of the esophagus. Further, one of these cases (A 30 115) showed extensive fibrosis of the circular muscular layer of the esophagus, indicating previous infection or the

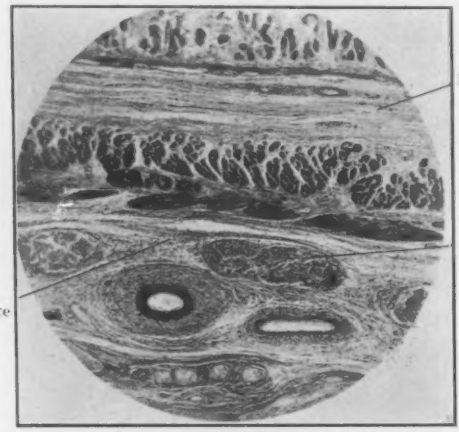



Fig. 13. A-30-143. Congenital syphilis and sepsis. Age 21 days. Section of the stomach. It shows a fine universal perigastric hemorrhage. There is fibrosis of the submucosal connective tissue. The vessels are filled with polymorphonuclear leukocytes.

action of toxines. The hemorrhages came from the capillaries. In my examination of the esophagus of babies and infants, hemorrhages into and around the esophagus are new findings. Except in one case, the baby with congenital syphilis, histological signs of infection could not be proved. It is well known that in cases of sepsis petechial hemorrhages are common in various parts of the body, especially in the skin. Taking the hint from the striking changes in the eclamptic baby, and in the baby who had congenital syphilis and died of sepsis, I feel that the probable cause of the hemorrhages in all the cases is a prebirth toxemia.

TWO CASES OF PYLORIC STENOSIS.

Before taking up the second main division of the paper, namely, the findings in the gall bladder and liver cases, I will record here the findings in two cases of pyloric stenosis. One baby was operated (Rhamstead operation). Both babies lived only a few days.

A 30 139. Pyloric stenosis. Marasmus. Rhamstead operation. *Microscopical Findings:* Esophagus at cardia. Great increase of subepithelial connective tissue. Periesophageal glands. No change in Auerbach's plexus. Stomach: Increase of subepithelial connective tissue. Great increase of the perigastric connective tissue.

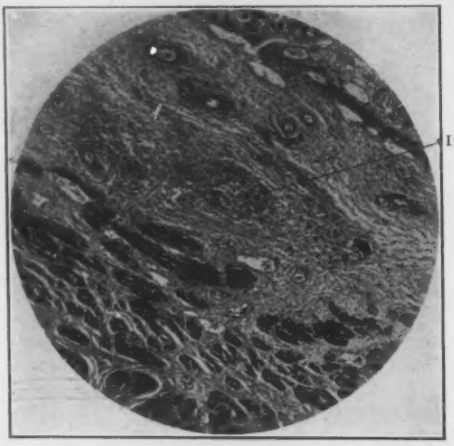



Fig. 14. A-30-143. Congenital syphilis and sepsis. Age 21 days. Stomach. High power. The section shows a marked infiltration of the submucosa with small lymphocytes. There is slight caseation.

A 30 153. Pyloric stenosis. Marasmus. *Microscopical Findings:* Esophagus above cardia. Marked periesophageal hemorrhage. Much periesophageal connective tissue. Periesophageal glands. The muscles of the lower esophagus in both of these cases were unusually sturdy. The most striking change, however, was the great increase of periesophageal connective tissue as shown by the Mallory stain.

The case of status lymphaticus in the tables may have caught the reader's eye so it will be disposed of at this point. The esophagus showed no changes. In a case of status lymphaticus described by the writer in a previous paper there was a great increase of the solitary follicles in the esophagus.

Fig. 15. A-30-143. Age 21 days. Congenital syphilis and sepsis. Section taken through the thick area of the stomach. Notice the change in epithelium. There is a slight change in epithelium in smooth area also. There is hemorrhage into the periconnective tissue. Dr. T. B. Mallory, Pathologist at the Massachusetts General Hospital, has seen but one other instance of this change in the mucosa of the stomach. See Fig. 16.

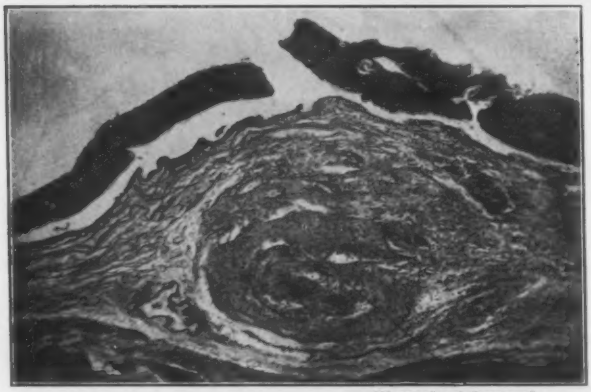



Fig. 16. McGregor No. 3. Male. Age 70 years. The patient died of cardiospasm or fibrosis of the terminal portion of the esophagus. The stomach was covered with placques. There were three small superficial ulcers. The photomicrograph shows the epithelium of the stomach changed in places to stratified squamous epithelium. There is a thrombosed vessel in the submucosa. The stomach was chronically infected. No stomach mucosa was found. This slide is introduced here because it shows the same change in the epithelium as is shown in Fig. 15 and because the change according to Dr. Mallory, is rare.

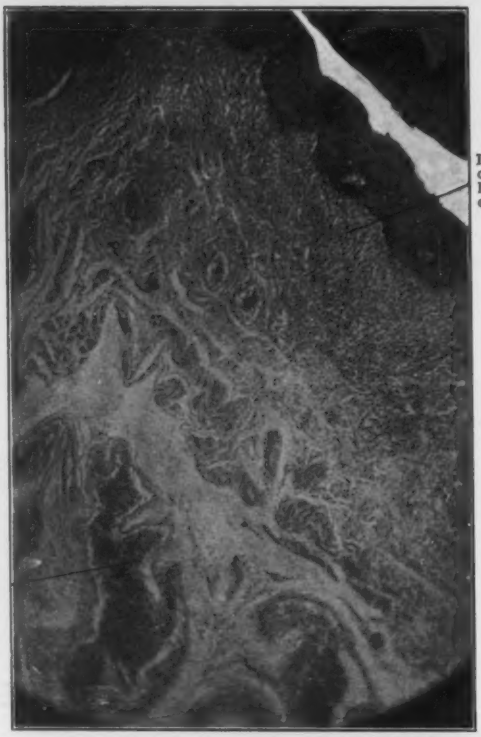



Fig. 17. A-30-160. Specimen of the esophagus from a case of myeloid leukemia. The vessels are filled with small lymphocytes. There is a subepithelial infiltration of small lymphocytes.

In the 43 specimens of the terminal portion of the adult esophagus, 13 proved of interest, five mildly so and eight of great interest.

There were six cases of appendicitis with general peritonitis. So far only two of these specimens have been run through.

A 30 131. Ruptured appendix and general peritonitis. *Microscopical Findings:* Many small lymphocytes among the glands of the esophagus.

A 30 154. Acute appendix. Operation. Bronchopnuemonia. *Microscopical Findings:* Many small lymphocytes and polymorphonuclear leukocytes among the glands of the esophagus.

A 30 167. Aortic aneurism and syphilis. Macroscopically the terminal portion of the esophagus seemed greatly thickened. The

microscopical sections showed this to be due to large muscle bundles in the longitudinal layer. Auerbach's plexus showed no change.

30903 M. G. H. Myeloid leukemia. Postoperative hemorrhage. Bronchopneumonia. *Microscopical Findings:* Esophagus: Subepithelial infiltration with small lymphocytes. Vessels filled with lymphocytes, mostly small. Auerbach's plexus showed an occasional slight infiltration with small lymphocytes.

A 30 160. Myeloid leukemia. *Microscopical Findings:* Esophagus: Vessels filled with small lymphocytes. Auerbach's plexus showed no change (see Fig. 17).

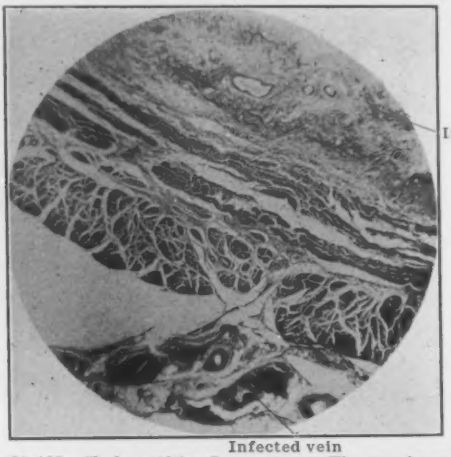



Fig. 18. A-30-135. Cholecystitis. Low power. The specimen shows many plasma cells under the epithelium and in the submucosa, indicating chronic infection. The periesophageal vessels are nearly thrombosed. See Fig. 19.

A 30 166. Diabetes mellitus. *Microscopical Findings:* Fibrosis of the circular muscle layer of the terminal portion of the esophagus. Agglutination and fibrosis of muscle bundles. Auerbach's plexus showed no change.

A 30 164. *Pathological Diagnosis:* Degeneration of anterior horn of spinal cord. *Microscopical Examination:* Esophagus: Sections from two places were put through. In one set there was a marked thinning of the circular muscular layer. Auerbach's plexus, no change. Stomach: No change.

The description of the following seven cases constitute the second, the concluding and, perhaps, the most striking part of the paper.

The cases in this group consist of four of gall bladder infection, two of atrophic cirrhosis of the liver and one of multiple abscess of the lung.

A 30 135. Cholecystectomy. *Microscopical Findings:* Many plasma cells under the esophageal epithelium, indicating chronic infection (see Figs. 18 and 19).

A 30 8. Cholecystectomy. Terminal portion of the esophagus moderately dilated. Esophageal mucosa gone down to the beginning of the mucosa of the stomach. *Microscopical Findings:* Subepithelial infiltration with small lymphocytes. Many small lymphocytes in the vessels. These findings indicate chronic infection (see Fig. 20).

Fig. 19. A-30-135. Cholecystitis. High power. The periesophageal vessels show many polymorphonuclear leukocytes. The vein in the illustration is nearly thrombosed. See Fig. 18.

Payson. Cholecystitis and operation. Age 64 years. Ill five months; gall bladder symptoms. *Pathological Diagnosis:* Peritonitis, pancreatic abscess, chronic myocarditis. At the terminal portion of the esophagus, about an inch above the beginning of the stomach mucosa, a string-like ridge crossed transversely the right half of the esophagus and projected into the lumen. This proved to be a fibrous septum (see Figs. 21 and 22). *Microscopical Findings:* Marker fibrosis. Deficiency of the muscle layers at the point of the septum. Agglutination of muscle bundles (see Figs. 21 and 22).

A 30 116. Atrophic cirrhosis of the liver. *Microscopical Findings:* Esophagus: Greatly dilated capillaries and veins. Dilated ducts. Ducts surrounded with a pronounced ring of lymphocytes. In one ring hemorrhage. Ducts nearly filled with polymorphonuclear

leukocytes. These findings indicate a hindrance to drainage and infection (see Figs. 23, 24 and 25).

Mrs. L., age 60 years, referred by Dr. Walter Lane, Milton, Mass. Atrophic cirrhosis of liver. Fibrosis of the terminal portion of the esophagus. About 12 years ago this patient was referred to me by Dr. Lane for difficulty in swallowing (see Figs. 26 and 27).

The X-ray picture was that of a typical case of advanced cardiospasm. The terminal four inches of the esophagus bent sharply to the left and lay flat on a motionless diaphragm. Dilatation of the

Fig. 20. A-21-8. Specimen of the lower end of the esophagus from a patient who died after a gall bladder operation. On gross examination the lower part of the esophagus was moderately dilated. The epithelium was gone down to the beginning of the stomach mucosa. On microscopical examination there was nearly complete loss of epithelium. There were many plasma cells in the submucosa. In other words, the esophagus was chronically infected.

crural portion of the esophagus was attempted under ether anesthesia. It was very hard to find the opening into the stomach. The esophagoscope could not be advanced into the stomach. A certain amount of dilatation was accomplished with bougies. From my point of view the whole procedure was unsatisfactory. However, enough dilatation was accomplished so that the patient lived long in comparative comfort, choosing, of course, her food to suit her condition. Dr. Lane and I discussed the feasibility of anastomosing the lower

end of the dilated esophagus and the fundus of the stomach. Nothing came of this; and, as I said, the patient lived along until about six weeks ago, when she strangled to death. Dr. Lane was away and did not see her at this time. Through his co-operation an autopsy was obtained. Dr. Branch did the section.

Fig. 21. Payson. This patient died after cholecystectomy. Just at the junction of the esophageal mucous membrane and the stomach mucosa a ridge-like septum extended from the right, half-way across the lumen of the esophagus. The illustration is to show the ridge. The esophageal muscles are seen to end, for the moment, at the web. See Fig. 22.

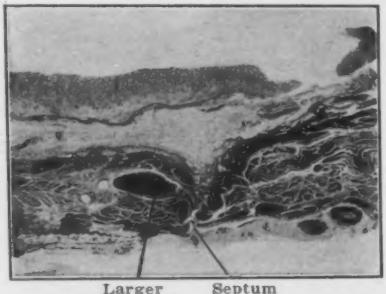



Fig. 22. Payson. Specimen showing a septum of the esophagus. X7. This patient died after cholecystectomy. The microscopical section shows that both muscular layers of the esophagus end in a point at the place where the septum occurs.

On exposing the esophagus it was found to be tremendously dilated, measuring in transverse diameter nearly six inches. It was full of fluid to the cricoid. The left half of the diaphragm was very low. The terminal portion of the esophagus lay flat on the diaphragm and was so large that the pathologist at first thought he was dealing with a diaphragmatic hernia of the stomach. The liver was small and had two or three large scars on its surface. The terminal inch

of the esophagus was tubular, soft and was not thickened. It admitted the tip of the little finger easily. The upper part of the esophagus was thin. The esophagus above the tubular portion was thickened to about twice the normal. This enlargement extended upward for about the lower third of the esophagus, at which point the esophageal wall thinned out. The fundus of the gall bladder was covered with many adhesions. So was the Spigelian lobe of the liver. Two band-like adhesions crossed the front face of the tubular portion of the esophagus and many adhesions passed behind it. The gall bladder was thickened (see Figs. 28, 29, 30, 31, 32 and 33).

Fig. 23. A-30-116. Atrophic cirrhosis of the liver. Low power. The section shows that the ducts are dilated and infected and often surrounded by a ring of small lymphocytes. The ducts contain many polymorphonuclear leukocytes. These findings indicate venous obstruction, and infection.

*Microscopical Findings:* The thin upper portion of the esophagus showed a very thin longitudinal layer. In places this layer was entirely wanting. In the lower part of the esophagus just above the tubular portion, where to the eye the esophagus was about twice as thick as normal, both muscular layers of the esophagus were thickened, especially the circular layer. The connective tissue between the muscular layers and about the esophagus was much increased, and in the circular layer, what I have never seen before to such an extent, the muscle bundles, in many places, were nearly one-half fibrous tissue (Mallory stain).

The tubular portion, that is, the part surrounded by the adhesions, showed great atrophy of both muscular layers. The muscle bundles showed fibrosis. The esophagus at this point was almost a connective tissue tube.

The stomach wall just below the tubular portion of the esophagus showed an even greater atrophy of its muscles (see Fig. 28). The gall bladder showed marked fibrosis, and hyaline degeneration of the

Fig. 24. A-30-116. Atrophic cirrhosis of the liver. High power. The section shows that both the veins and the ducts are dilated and infected. The ducts are surrounded by a ring of small lymphocytes. These findings indicate venous obstruction, and infection.

muscle coat. The liver showed the picture of atrophic cirrhosis.

Here then is a case of cardiospasm due to constricting adhesions about the terminal portion of the esophagus, caused by infection from the gall bladder. The continued infection produced a great atrophy of the muscles at the terminal portion of the esophagus and even greater atrophy of the muscles of the first part of the stomach. Naturally Auerbach's plexus had practically disappeared from the

lower part of the esophagus. I believe that it was the infection also which caused the extensive fibrosis of the circular layer of the esophagus.

The four cases just described show that disease of the gall bladder or of the liver can be associated with pathological changes in the terminal portion of the esophagus. The last case, I believe, illus-

Fig. 25. A-3-116. Atrophic cirrhosis of the liver. High power. The section shows dilated capillaries and veins. The ducts are dilated and infected. They are surrounded by a ring of small lymphocytes. There is hemorrhage about one duct. There is fibrosis of the circular muscular layer.

trates one type of cardiospasm; that is, the type where infection causes periesophageal adhesions with constriction of the terminal portion and dilatation and hypertrophy of the esophagus above the constriction. With the dilatation of the esophagus comes a backward bend at the terminal portion, which makes the obstruction practically permanent. The terminal portion of the esophagus finally becomes

disorganized from constriction and chronic sepsis. The esophagus above the tubular portion, probably at first hypertrophies, then from continued infection within and without the muscle fibres becomes fibrosed and then atrophied.

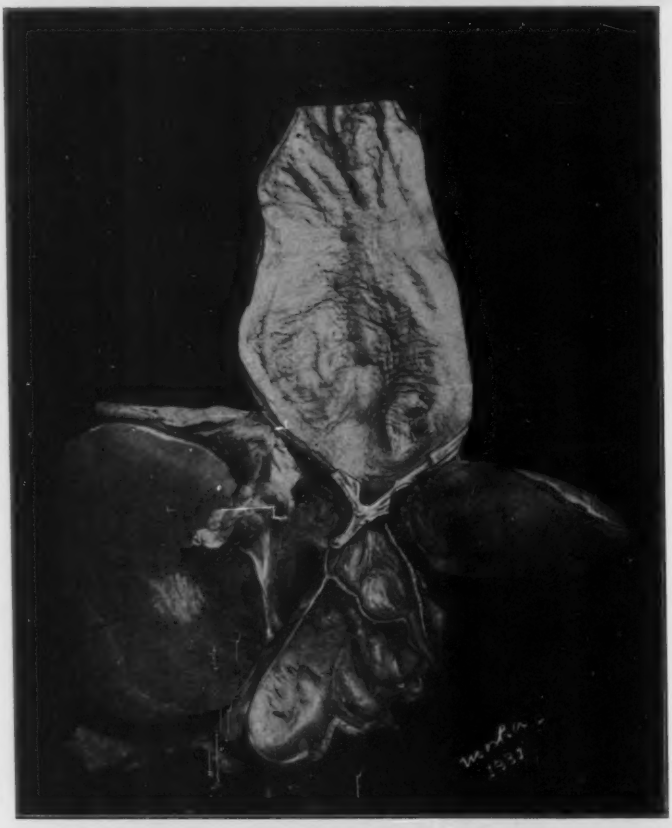


Fig. 26. Lawrence. Retouched photograph of the stomach, gall bladder and liver. The upper part of the stomach has been opened. The illustration shows that the terminal portion of the esophagus is surrounded by band-like adhesions. The esophagus is greatly dilated except at the terminal constricted portion. The liver is scarred and atrophied. This patient had cardiospasm for 12 years.

The two commonest diseases of the abdomen are infection of the gall bladder and of the appendix. Therefore, if fibrosis in and about the esophagus occurs, and these cases show that it does, the infec-

tion which is the forerunner of the fibrosis and its cause should often originate in the gall bladder or liver.

In my own mind I have summed the whole matter of the cause of cardiospasm up as follows: 75 per cent of the cause is periesophageal fibrosis at the terminal portion of the esophagus caused

Fig. 27. Lawrence. The esophagus, stomach and liver seen from behind. The illustration shows that the terminal portion of the esophagus is overlain by many adhesions. Microscopically the liver is shown to be atrophied and the gall bladder chronically infected and fibrosed.

by neighborhood infection, or fibrosis of the terminal portion of the esophagus; 20 per cent is backward bend of the terminal portion; 5 per cent perhaps is spasm, aiding and abetting the two chief causes. As to the part played by infection and fibrosis of Auerbach's plexus in cardiospasm I feel that involvement of the plexus is a

Fig. 28. Lawrence. Section of the upper part of the esophagus. X20. The section shows that the longitudinal layer of muscles has nearly disappeared.

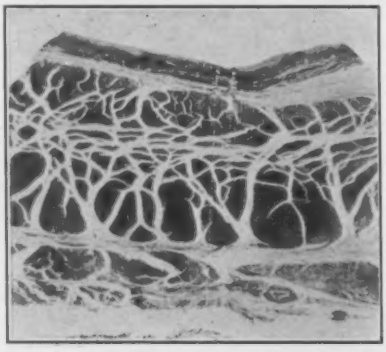


Fig. 29. Lawrence. Lower third of esophagus. X20. The section shows great thickening of the circular layer. There is much fibrosis of the muscular bundles. The dark areas are patches of fibrosis.

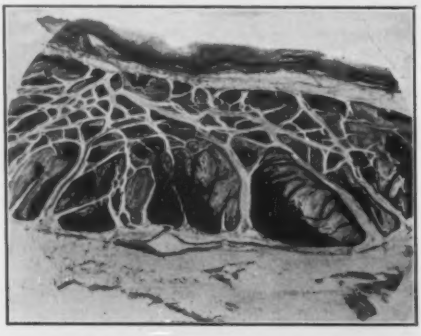


Fig. 30. Lawrence. Lower third. X20. Retouched to show fibrosis. The dark areas are due to fibrosis. The longitudinal layer in this section is wanting. The circular layer is greatly thickened, as well as fibrosed. This section is taken from the thickest part of the esophagus; that is, just above the tubular portion.

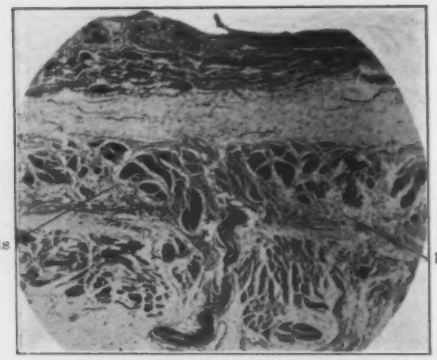



Fig. 31. Lawrence. Tubular area of esophagus X20. The section shows that both muscular layers, especially the circular, are largely replaced by fibrous tissue. Auerbach's plexus is much disorganized.

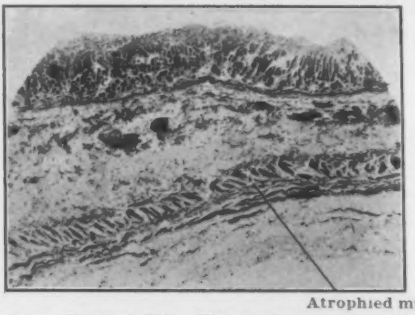



Fig. 32. Lawrence. Stomach X20. The section shows that the muscles of the stomach are very much atrophied. The section of the stomach comes from just below the constricted tubular portion of the esophagus.

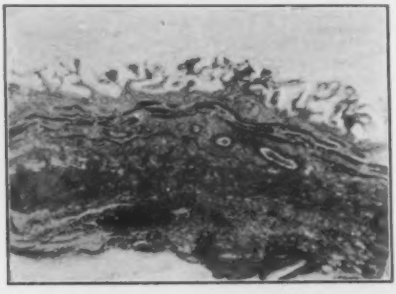


Fig. 33. Lawrence. Gall bladder X20. The section shows that the gall bladder is much disorganized. The muscles are largely replaced by fibrous tissue.

secondary and not a primary affair. Of course, to the degree that changes occur in the plexus, the motor function of the muscles of the esophagus is interfered with. Theoretically, infection might involve the plexus first, but I have examined its condition in a good many acute cases and have found that it is not easily involved.

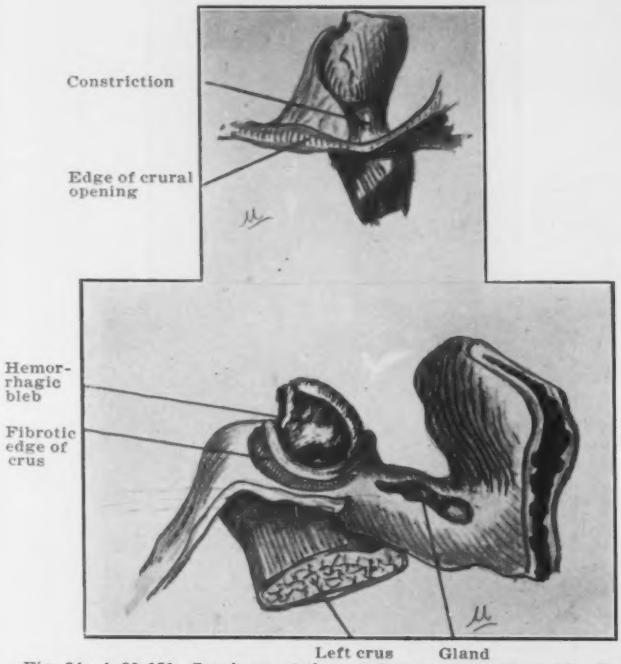



Fig. 34. A-30-151. Specimen of the esophagus of a patient who died after an operation for acute cholecystitis. The upper figure in the illustration shows a constriction in the esophagus caused by a fibrosis of the crural ring. The lower figure shows a bleb on the external wall of the esophagus. This measured three-quarters of an inch in height, and half an inch in width. It was filled with blood. In this instance the writer believes that a subepithelial hemorrhage broke into the esophagus and a hemorrhage in the body of the esophagus broke externally, practically rupturing the esophagus.

In the last three cases I return to the first subject treated in the paper, namely, periesophageal hemorrhage and hemorrhage into the esophagus. The three specimens about to be described, however, are not specimens of the esophagus from babies dead prematurely or at term, but specimens of the adult esophagus.

A. Diabetes mellitus. The specimen consisted of the terminal four inches of the esophagus and the upper two inches of the stomach. On cross-section of the esophagus in three places gross examination showed numerous subepithelial hemorrhages. The stomach showed even more extensive subepithelial hemorrhage. *Microscopical Exami-*

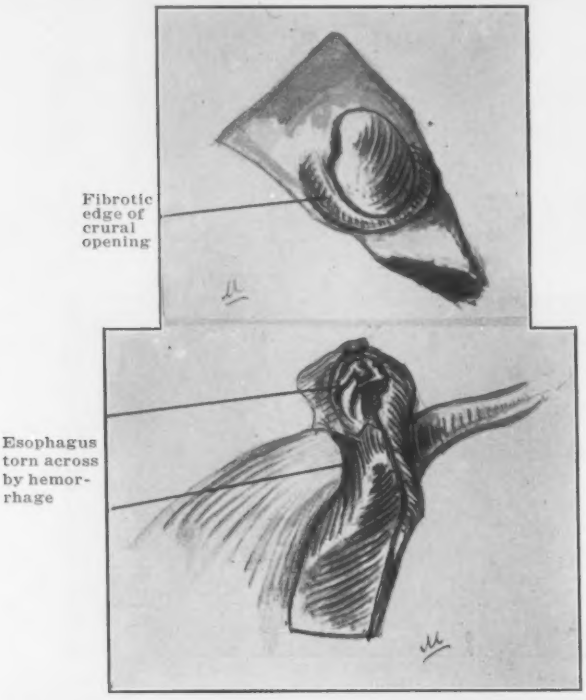



Fig. 35. A-30-151. Specimen of the esophagus from a patient who died after an operation for acute cholecystitis. The upper figure shows that the facial edge of the crural opening is fibrosed for nearly two-thirds of its circumference. The lower figure shows the constriction in the esophagus caused by the fibrosed crural ring. It shows also the esophagus practically torn across by hemorrhage.

*nation:* The appearance of subepithelial hemorrhages in the gross specimen proved to be due to dilated capillaries.

A 30 151. Acute cholecystitis. Icterus. Operation, drainage of gall bladder. Age 65. Male, white. *Previous History:* Fifteen months before entering the hospital, patient fell from a staging, striking on the chest and abdomen. He has not worked since. For

one month has had loss of strength, jaundice and clay stools. X-ray film shows pathological gall bladder. *Family History:* Negative. *Past History:* Except as above, negative.

After operation patient vomited almost all food taken by mouth. He gradually grew weaker and died on the eighteenth day.

*Microscopical Findings:* Fibrosis of two-thirds of the facial edge of the crural opening. Narrow and rigid crural opening with marked

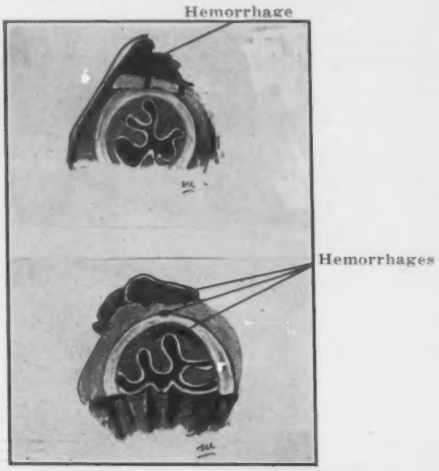



Fig. 36. Specimens of the esophagus from a case of multiple abscess of the lung. The patient from whom these specimens were obtained showed multiple miliary abscesses of the lung. The specimens are from the lower third of the esophagus. On cross-section a large periesophageal hemorrhage was found, and there were spots of smaller hemorrhage in the esophagus itself. Microscopic examination showed that what appeared to be hemorrhage under the epithelium was due to greatly dilated capillaries. It is common to find such apparent hemorrhages under the epithelium of the stomach. They were present in this case. Microscopic examination showed them to be due, as in the esophagus, to dilated capillaries. In one series of sections from this esophagus, both layers of the esophageal muscles were much fibrosed, showing that it had been infected from the lungs. No characteristic signs of tuberculosis, however, were found.

constriction on the terminal portion of the esophagus (see Fig. 35). Blue-black bleb above the crural opening and the constricted part of the esophagus. The bleb extended half way around the circumference of the esophagus and up the external wall of the esophagus three-quarters of an inch. A quarter of an inch above the bleb the esophagus ended in a number of jagged finger-like points. The lumen of the esophagus was filled with blood. The bleb was filled

with blood. The specimen ended at the finger-like projections as if the esophagus had been torn off here during removal. A cross-section showed the hemorrhage had disintegrated one-half of the wall of the esophagus at the top of the bleb.

Here again is a case of infection and a gall bladder case associated with hemorrhage, and one large enough to perforate the esophagus (see Figs. 34 and 35). The hemorrhage extended in the sheath of the esophagus down to the stomach.

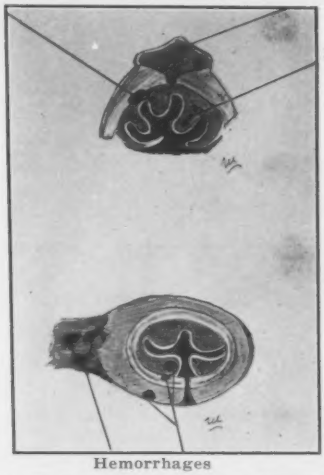



Fig. 37. Specimens of the esophagus from a case of multiple abscess of the lung. In the illustration the upper figure shows a periesophageal hemorrhage and dilatation of the subepithelial vessels. The lower figure shows three small periesophageal hemorrhages. The writer has a recent specimen—not figured—from the esophagus of a patient who died of a decompensated heat lesion. In the lower third of the esophagus for a distance of an inch the mucosa of the esophagus was covered with blood. There was extensive confluent subepithelial hemorrhage. Microscopic examination showed greatly dilated subepithelial vessels. In this case the blood had broken through to the surface.

A 31 16. White, female. Age 56 years. *Pathological Diagnosis:* Diabetes mellitus. Bilateral pneumonia. Bilateral multiple milliary abscesses. Healed apical tuberculosis.

*Clinical History: Present Illness:* Has had diabetes for the past two years, and has been under treatment for it. Eight days ago patient fell on icy sidewalk. A few days later developed a slight cough, which has gradually grown worse.

*Physical Examination:* Wassermann negative. X-ray, two large areas of consolidation at both bases of the lungs. Scattered areas of infiltration over both lungs.

*Clinical Progress:* Patient became progressively worse, went into coma and died without regaining consciousness in 25 hours.

*Multiple Abscess of the Lung:* The specimen which came to me for examination was the lower four inches of the esophagus and the upper two inches of the stomach.

*Gross Findings:* On the external wall of the esophagus at about the middle of the specimen there was a hemorrhage bleb half an inch in its vertical dimension and a quarter of an inch in its transverse. On cross-section of the bleb the hemorrhage was seen to extend through the longitudinal layer; that is, half-way through the esophageal wall. Four cross-sections were made an eighth of an inch apart. They showed hemorrhage in each compartment of the esophagus. In one section there were three such hemorrhages; in another, two. One section showed three hemorrhages in the sheath of the esophagus.

These two cases show that periesophageal hemorrhage and hemorrhage into the musculature of the adult esophagus occurs. The hemorrhage may even cause perforation of the esophagus. It is noteworthy that both the patients from whom these specimens were taken suffered from infection. In one it was acute—the gall bladder case; in the other, chronic—the case of multiple lung abscess. In periesophageal hemorrhage, such as occurs in these two cases, we have a cause of the unexplained cases of periesophageal fibrosis below the cricoid cartilage, such a narrowing of the esophagus being due to the organization of a periesophageal hemorrhage which was not sufficiently large to perforate the esophagus (see Figs. 36 and 37).

## SUMMARY.

The esophagus may be infected before birth.

Periesophageal hemorrhage has been found in premature babies born dead.

In babies dying with hemorrhagic disease of the newborn, periesophageal hemorrhage has been found, as well as subepithelial hemorrhage in the esophagus and the stomach.

Extensive intramuscular and periesophageal hemorrhage has been found in a baby which lived 36 hours and died of intracranial hem-

orrhage; in a baby whose mother had toxemia of pregnancy, and in a baby born with congenital syphilis, which died of sepsis from ulcerative tonsillitis and pharyngitis.

The hemorrhages were found to come from the smallest of the capillaries.

The hemorrhage in babies dead at birth is probably due to prebirth toxemia.

Hemorrhagic disease of the newborn is probably due to prebirth toxemia.

Infection of the gall bladder, one the commonest of abdominal diseases, often is associated with infection of the terminal portion of the esophagus. By causing adhesions it is one cause of cardiospasm. Very extensive intramuscular and periesophageal hemorrhage has been found associated with acute cholecystitis and jaundice.

Associated with fibrosis of the facial edge of the crura, narrowing of the crural opening and constriction of the esophagus has been found.

Two cases of peri- and intraesophageal hemorrhage have been found in adults, one associated with acute infection (cholecystitis), and the other with chronic infection (multiple lung abscess).

828 Beacon Street.

# PRELIMINARY REPORT OF A STUDY OF THE PHILOGENESIS AND ONTOGENESIS OF THE FOOTPLATE OF THE STAPES, WITH A THEORY OF OTOSCLEROSIS.*

DR. LOUIS K. GUGGENHEIM, St. Louis.

In studying the ear regions of embryos the writer has found that the footplate of the stapes, instead of being formed by the stapedial ring (derivative of Reichert's cartilage) as heretofore reported, is in reality derived from the labyrinthine capsule. This new fact was reported before the Triological Society in June of this year. In discussing the paper (The Development of the Organ of Hearing), Arthur Proetz commented upon the discovery of the origin of the footplate as being the most interesting and important part of the presentation. The thought, "like to like," occurred to him in connection with the relation of the capsule derived footplate to otosclerosis. This suggestion led the writer to study the organ of hearing from the Myxine glutinosa, the lowest vertebrate, to man. The phylogenetic investigation shows just when the oval window first makes its appearance, the development of the tympanic contents, etc.

Next a careful investigation of the ontogenesis of the oval window and stapes was made.

In the lower vertebrates there is no oval window, the labyrinth being entirely closed off. The first vertebrate to show an oval window comparable to man is the Acanthius vulgaris. In man and the higher vertebrates we note at five weeks (see Fig. 1) a membranous labyrinth surrounded by condensed mesenchyme (precartilage) and entirely shut off from the outside; no sign of a labyrinth window having made its appearance. At seven weeks (see Fig. 2) the labyrinth shows dedifferentiation of precartilage into syncytial reticulum. Perichondrium has been differentiated. The external wall of the vestibular division shows a deep indentation where Reichert's cartilage forms the stapedial ring but no sign of a window is present; the perichondrium being plainly continuous over the entire external surface of the labyrinth. At nine weeks (see Fig. 3) Reichert's cartilage has formed a complete ring around the stapedial artery, the ring forming a deep indentation in the external wall of the capsule. The internal segment of the ring now occupies the position

*From the Oscar Johnson Institute, Washington University Medical School, St. Louis.

Fig. 1. Rat (human age, 5 weeks). 1. Labyrinth capsule (precartilage). 2. Constriction for division of auditory vesicle into utriculus and sacculus. 3. Semicircular canal. 4. Spiral ganglion. 5. Utriculus. 6. Sacculus. 7. Cochlea.

Fig. 2. Human, 7 weeks. 1. Stapes. 2. Labyrinth capsule (precartilage). 3. Perichondrium. 4. Cochlea. 5. Dedifferentiation of precartilage into syncytial reticulum. 6. Semicircular canal. 7. Tympanum (mesenchyme filled).

Fig. 3. Rat (human age, 9 weeks). 1. Labyrinth capsule (precartilage). 2. Perichondrium. 3. Stapedial ring. 4. Cochlea. 5. Myelencephalon. 6. Reichert's cartilage.

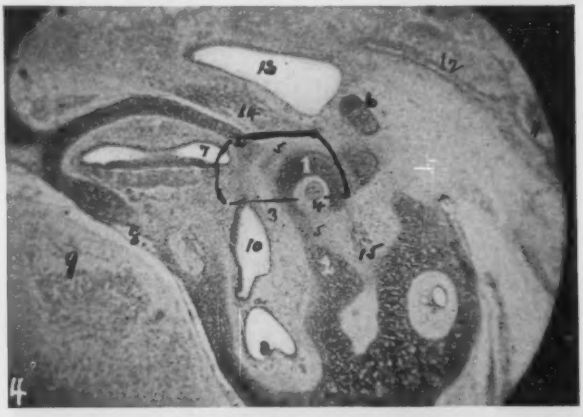



Fig. 4. Rat (human age, 11 weeks). 1. Stapedial ring. 2. Labyrinth capsule (cartilage). 3. Syncytial reticulum in cisterna perilymphatica. 4. Stapedial artery. 5. Condensation in precartilage curtain of oval window to form annular ligament. 6. Reichert's cartilage. 7. Cochlea. 8. Internal auditory meatus. 9. Myelencephalon. 10. Utriculus. 11. External auditory canal. 12. Drum membrane (ectodermal and entodermal layers). 13. Eustachian tube. 14. Tensor tympani muscle. 15. Stapedial muscle.

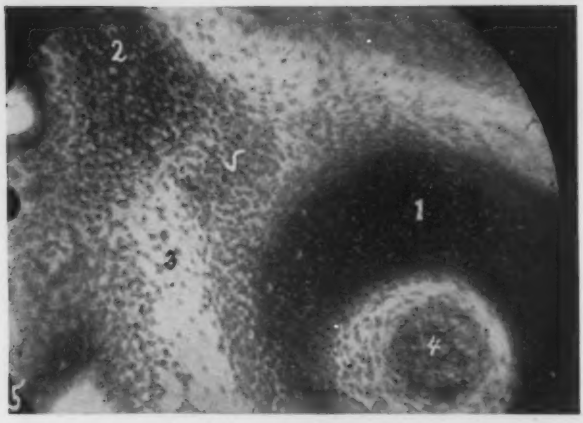



Fig. 5. Rat (human age, 11 weeks). Outlined area in Fig. 4. 1. Stapedial ring (cartilage). 2. Labyrinth capsule (cartilage). 3. Syncytial reticulum in cisterna perilymphatica. 4. Stapedial artery. 5. Curtain covering oval window (condensed mesenchyme—precartilage).

Fig. 6. Rat (human age, 13 weeks). 1. Cochlea. 2. Cisterna perilypmhatica. 3. Stapedial artery. 4. Labyrinth capsule (cartilage). 5. Stapedial ring. 6. Reichert's cartilage. 7. Tensor tympani muscle. 8. Stapedial muscle. 9. and 10. Condensations in curtain to form annular ligament. 11. Eustachian tube.

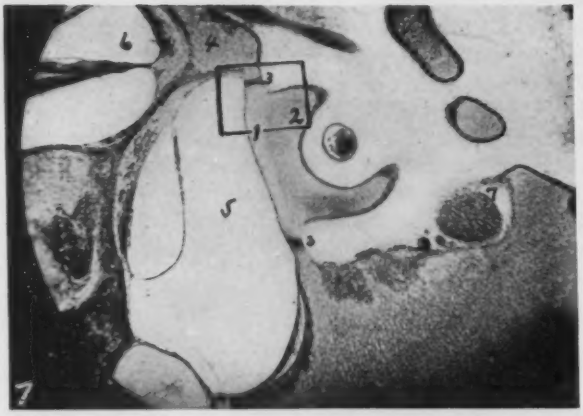



Fig. 7. Rat (human age, 16 weeks). 1. Footplate of stapes (cartilage). 2. Anterior crus of stapes. 3. Annular ligament. 4. Capsule of labyrinth (bone and cartilage). 5. Cysterna perilymphatica (free of syncytial reticulum). 6. Cochlea. 7. Facial nerve.

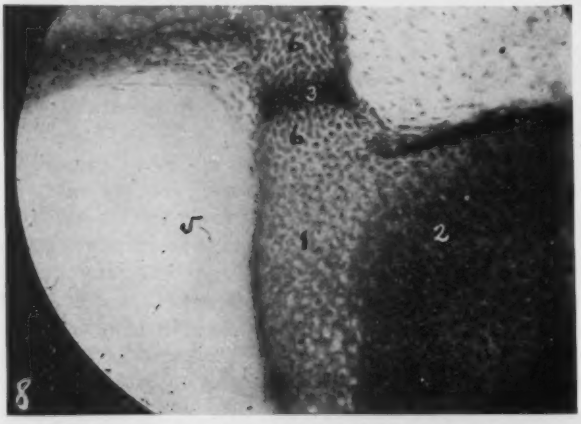



Fig. 8. Rat (human age, 16 weeks). Outlined in Fig. 7. 1. Footplate of stapes (cartilage). 2. Anterior crus of stapes. 3. Annular ligament. 4. Labyrinth capsule (cartilage and bone). 5. Cysterna perilymphatica. 6. Condensed mesenchyme.

of the future footplate. It is this embryologic picture which has probably led all embryologists heretofore to ascribe to the Reichert ring the rôle of footplate producer. Looking closely with a magnifying glass we see that the perichondrium is still unbroken under the ring. At 11 weeks (see Fig. 4) the entire labyrinthine capsule has changed from precartilage into cartilage, except at the internal auditory meatus and at the region subjacent to Reichert's ring. At the latter location we note that the capsular cartilage stops rather abruptly and that the potential oval window is now covered by a mass of condensed mesenchyme cells with a moderate amount of connective

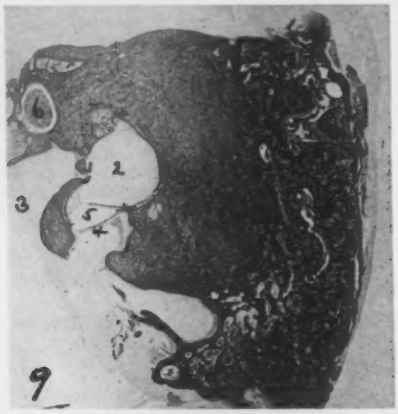



Fig. 9. Human (adult). 1. Footplate of stapes with annular ligament. 2. Cysterna perilymphatica. 3. Tympanum. 4. Membrana tympani secondaria. 5. Scala tympani of cochlea. 6. Facial nerve.

tissue. The stapedial ring is now of pure cartilage, totally unlike the condensed mesenchyme of the oval window covering. A higher magnification of Fig. 4 (see Fig. 5) shows the cartilaginous stapedial ring surrounding the stapedial artery. Subjacent to the ring we see the condensed mesenchyme passing over to the labyrinthine cartilage. The condensed mesenchyme cells are sharply delimited from the cartilage cells of the ring, whereas they blend with the cartilage cells of the capsule so plainly that there can be no doubt that they are part and parcel of the capsule itself. In a word, when the capsule undergoes differentiation from precartilage to cartilage, the future oval window area remains at the precartilage stage for a while. Also note that the marked indentation of the capsule by the ring has decreased. At this stage is further seen an additional degree of condensation at two points in the photomicrograph (see Fig. 4), the

site of the future annular ligament. Note that these two points are far anterior and posterior to the limits of the ring.

At 13 weeks (see Fig. 6) the segment of the ring adjacent to the oval window covering has become flattened to accommodate itself to the latter. The covering of the oval wind now shows many cartilage cells and the two points of greater condensation (annular ligament) are more definite than in the last figure. In the photomicrograph it is quite apparent that the footplate and the stapedial ring are two distinct structures; they do not as yet even fit together.

At 16 weeks (see Fig. 7) the annular ligament has assumed almost its adult form. The ring and the footplate now fit as if made together but one sees quite plainly that between the two is a marked difference of texture. Under higher magnification the footplate and the labyrinth capsule are now identical (see Fig. 8) and distinctly different from the stapedial ring division of the stapes. All three structures are now cartilaginous. On either side of the annular ligament, anterior to the ring one sees persisting condensed mesenchyme. This is the site of predilection for the beginning of otosclerosis. These condensed mesenchyme cells are still not "fixed" and are pluripotential. They may remain as they are; they may resolve into a syncytial reticulum which may resorb; they may differentiate into cartilage; they may be the ground work for fibre bone (otosclerosis). It would seem, almost, as if Nature, uncertain of the advisability of admitting extraneous sound (the lowest vertebrates have only an organ of equilibrium) retains this means of closing the window if she sees fit. Brühl's theory that the pull of the tensor tympani muscle acts as an irritant to the anterior segment of the annular ligament and thus initiates the new bone formation in otoscelrosis would seem to be refuted by the fact that there are hundreds of places in the body where muscle pull is greater than at others but no new bone formation results.

We know nothing of the cause of otosclerosis other than its hereditary characteristic. Nothing of cause then is known except that in certain individuals the chromosomes transmit a something which causes the oval window to close with bone (the involvement of the remainder of the capsule will be considered in the complete paper). The typical site is just anterior to the footplate of the stapes. The new bone is a fibre or web bone. It obliterates the oval window by replacing the footplate and annular ligament. The labyrinth becomes shut off as far as its primitive portion (vestibular apparatus) is concerned; just as it is shut off in the lower vertebrates. May we not, therefore, look upon otosclerosis as a regression rather than a disease process?

Oscar Johnson Institute.

# PREVENTION OF ACUTE SURGICAL MASTOIDITIS.*

DR. ARTHUR WAGERS, Philadelphia.

By the acute surgical mastoid is meant mastoid infection which must be operated upon to effect a cure. From this statement it may be inferred that there are cases of acute mastoiditis, which, if accorded sufficiently early and proper attention, can be cured without resort to operation. I believe this number to be large enough to merit serious consideration.

Mastoidectomy is to be avoided, if possible, for the following reasons:

*1.* Patients and their families have a justifiable dread of a mastoid operation.

*2.* The possible complications of acute mastoiditis—sinus thrombosis, brain abscess and meningitis, even though they receive the most skillful surgical attention, are all serious complications, fraught with danger to the patient's life.

*3.* The expense of an operation, including several days in the hospital, loss of time from one's vocation, nursing service and the surgeon's fee, create a financial obligation formidable to contemplate in many instances.

If we grant the truth of these statements, it is not difficult to understand the desirability of preventing as far as possible the worry, the danger and the expense attendant upon the development of the acute surgical mastoid.

Attempting to prevent this, we must direct our attention to the origin of acute mastoiditis.

Very few mastoids become infected from the blood stream or lymphatic circulation, and the cases are so rare as to be practically negligible. Most of the cases seen are complications of acute middle ear suppuration and the usual route of infection is by way of the Eustachian tube. It is a matter of common knowledge that middle ear infection is prone to occur during attacks of measles, scarlet fever, chickenpox, pneumonia, acute tonsillitis, influenza, from swimming etc. It is, therefore, highly important that the possibility of infection from these sources be borne in mind and the ears be kept under close observation, even without ear symptoms, in the case of

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*Read before the Philadelphia Laryngological Society, Tuesday, Feb. 3, 1931.

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young children and infants, and at the first sign of discomfort on the part of older children and adults.

In any case of acute middle ear infection which is seen sufficiently early, that is, before there is marked redness of the membrana tympani, it may be possible to abort the infection. The patient, if not already in bed, should be put to bed and given a laxative. A cotton tampon saturated with 10 per cent phenol in glycerin is placed in contact with the membrana tympani. The patient should be seen again in from 10 to 14 hours. If the laxative given has been effective and if the redness of the membrana tympani has disappeared, one concludes that these simple abortive measures are producing the desired result, and it is quite probable that within a few more hours the danger of suppurative otitis media will have passed. If, at this time, the membrana tympani shows the same or increasing redness, but no bulging, one might be justified in prolonging the phenol in glycerin tampon for another period of a few hours. By this time the appearance of the membrana tympani and the subjective symptoms of pain should be better or worse. If the membrana tympani now shows bulging in any portion, free incision and drainage of the accumulated pus is clearly indicated and should be carried out without further delay. I have suggested waiting for the appearance of bulging before incision of the membrana tympani. This is the plan usually followed, except in cases showing marked redness of the drum during an attack of scarlet fever, measles or other exanthemata. Experience has shown that nothing is to be gained by abortive measures in these cases and incision is resorted to promptly, even though there be no bulging. What we do with the acute otitis media at this stage determines in many cases whether or not a surgical mastoid develops. The longer a purulent exudate remains in the tympanic cavity under pressure, the greater the danger of mastoid involvement; therefore the urgency of free drainage at the earliest sign of accumulation which produces bulging of the drum membrane. Another reason for early incision rather than waiting for spontaneous rupture is that before the latter occurs the weakest area in the drum has become nearly, if not quite, necrotic and when the pressure finally becomes sufficient to produce the rupture, the edge of the perforation is so devitalized that it sloughs off, leaving in most cases a permanent opening in the membrane. Such a result is undesirable, for the reason that another and permanent route for infection is opened to the middle ear and the membrana tympani cannot perform its normal function as efficiently as before. It happens often, of course, that spontaneous rupture has already taken place when the patient is first seen. In such cases it is the duty of

the physician to determine whether or not the rupture is sufficiently large to provide adequate drainage; if not, the opening should be extended by free incision.

Having now a completely developed middle ear infection and drainage provided, one of three terminations would naturally come about were the patient to receive no further treatment: either the ear would continue discharging for a brief time and cease spontaneously, or continue discharging indefinitely as a chronic suppurative otitis media, or an acute suppurative mastoiditis might develop. It is the latter condition we now seek to prevent. In an attempt to accomplish this, we treat the suppurative otitis media daily. If the external auditory canal is filled with pus, this is removed by means of a suitable glass tube connected with a suction pump. This accumulation of pus being removed from the canal, a larger, cone-shaped tip is inserted closely into the entrance of the canal and mass suction applied to remove the pus contained in the tympanic cavity. Care must be taken that this suction is not too strong. It requires very little negative pressure to remove the pus and greater pressure may produce a hematoma in the canal wall and also bring about sufficient congestion of the mucous membrane of the tympanic cavity to defeat its purpose by blocking the drainage through the Eustachian tube, and even through the incision in the membrana tympani. There is often a tendency toward too rapid healing of the incision and when this occurs and it is evident there is a greater amount of pus to be evacuated than can pass out freely, the incision must be extended with the knife and the daily treatment continued.

After the middle ear has been thoroughly emptied of the contained pus, local applications of various sorts may be employed. A moderately strong solution of alcohol and boric acid may be used on a cotton-wound applicator for further cleansing of the canal, after which wet or dry applications are made, according to the indications and the individual preference of the attending physician.

This in brief is the plan of treatment which has given me the most satisfactory results in preventing the acute surgical mastoid.

In a paper which I presented before the Pennsylvania State Medical Society in 1924, mention was made of approximately 75 cases of acute suppurative otitis media which had been seen and treated during a three-year period. Only one of these had become chronic and none had developed a surgical mastoid. All these cases were seen early and for the most part received daily treatment until cured. Their treatment was essentially that outlined above, except that they were not put to bed.

During the past 18 months I have been responsible for the treatment of ear conditions developing in the pediatric wards of Jefferson Hospital. During this time there have been no mastoids requiring operation, although there have been 51 cases of acute suppurative otitis media. These patients are seen and treated daily and the average duration of the discharge has been 17 days. It is not unreasonable to suppose that had treatment been neglected there would have developed among these children at least a few cases of acute mastoiditis requiring surgical interference.

So far I have considered cases examined in the earliest stage of acute otitis media and up to the appearance of bulging or spontaneous rupture of the membrana tympani and if our treatment of the middle ear infection has been successful there should be nothing more in the way of mastoid involvement than the congestive stage, which is commonly believed to be present in all cases of acute suppurative otitis media.

We have still to consider the case seen later on after perhaps three or four weeks of middle ear suppuration, without adequate drainage, or the case which is already developing symptoms of acute mastoiditis—pain over the antral area, at the tip of the mastoid, or over the point of exit from the skull of the emissary vein, along with an elevation of temperature. The hearing may become still more impaired and the aural discharge continues abundant and often pulsating as it comes from the tympanic cavity. Blood examination shows leukocytosis of some degree. With all these conditions present, but without drooping of the posterior superior wall of the canal, it is usually safe to make an attempt at abortion of the mastoiditis. The patient should be put to bed, given a strong cathartic, and it is quite probable that the membrana tympani will require incision to permit freer drainage. If it is felt that the mastoid involvement is in a sufficiently early stage, cold may be constantly applied for a period of 10 or 12 hours. If by that time the condition has improved, the cold may be continued until complete relief has been obtained. But if all signs and symptoms have steadily increased in severity in spite of the application of cold, the cold should be discontinued and hot saline irrigations substituted. A quart of hot normal salt solution, as hot as can be borne, is used every two hours, care being taken that the irrigator is held not more than three or four inches above the ear during the irrigation. If this line of treatment is successful, favorable results should become apparent within from 24 to 36 hours.

There can be no definite rules advanced for guidance at this time. One forms opinions from what is seen as the patient is observed

from time to time and the surgeon acts according to his best judgment. If he detects drooping of the canal wall, he will usually decide that operation is indicated. In some cases there may be no visible drooping but the thickened appearance of the drum membrane along with continued or increased pain in the mastoid area, continued elevation of temperature, and possibly postauricular swelling —these signs indicate that conservative treatment has failed and it is time to operate. Roentgenologic examination, although not previously mentioned, is of great assistance in doubtful cases.

In the so-called atypical mastoid, each case presents its own problems and these must be met according to the indications in the individual patient. Patients are seen, for instance, who do not complain of pain in any portion of the mastoid area, or there may be only a very slight increase in the number of leukocytes, or there may be no drooping of the external auditory canal wall and yet the mastoid may be filled with pus and granulation tissue. Such a condition of affairs may come about because the attending physician has not recognized the mastoid involvement in time to employ preventive measures.

Prevention of the surgical mastoid as I have indicated depends largely upon early recognition and treatment of ear conditions which are the forerunners of suppurative mastoiditis. That we may have such early recognition, we must impress upon the family physician, and, through him, the laity, the great danger which may arise not only to the sense of hearing but to life itself from neglect of immediate and proper attention to the ear at the first sign of discomfort.

In conclusion, I would stress the point that we cannot follow any fixed routine as to the time for operating on the mastoid. The idea of arbitrarily opening the mastoid in any case of acute suppurative otitis media that has continued discharging four or 10 or 20 days without any other indications is positively absurd. The rational thing to do is to keep the acute middle ear condition under constant observation and meet extension to the mastoid area if and when it occurs.

1429 Spruce Street.

# REPORT OF A CASE OF ACUTE MASTOIDITIS COMPLICATED BY INTERNAL HYDROCEPHALUS.*

DR. FRANK C. CARR, New York.

*History:* Baby R. C., age 7 months, was admitted to Dr. Stuart Craig's service at the New York Eye and Ear Infirmary on Oct. 3, 1930, with a subperiosteal abscess of the right mastoid.

*Chief Complaint:* Discharge from the right ear for one week. Swelling behind the right ear for one day.

*Family History:* Negative. *Past History:* Normal breast-fed infant. He has had continuous head colds since one month of age. The left ear discharged for two weeks when he was 4 months of age.

*Present Illness:* The right eardrum ruptured spontaneously a week ago. Two days ago there was a scant mucopurulent discharge, the drum was bulging and reddened and there was sagging of the superior wall, with marked mastoid tenderness. A wide incision was made in the drum membrane and only blood obtained. The next day the myringotomy wound was closed and the drum was again reddened and bulging. This was due to dense granulations filling the middle ear. The patient was then admitted to the hospital.

*Operation:* On Oct. 3 a right simple mastoidectomy was done. The entire mastoid cavity was practically excavated by the diseased process. A small exposure of the dura was made in the middle fossa at the time of operation. The child had an uneventful convalescence and was discharged from the hospital on Oct. 9 with the posterior wound closed by first intention and a slight discharge from the ear canal.

*Laboratory Work,* Oct. 3: Aural smear shows streptococci. Blood examination: Hemoglobin, 59 per cent; R. B. C., 3,200,000; W. B. C., 8,200; polys., 53 per cent; small lymphs., 41 per cent; large lymphs., 5 per cent.

The child was dressed every other day for 25 days. During this time the purulent discharge from the ear canal became more profuse.

On Nov. 3, a month after the original mastoidectomy, the child was readmitted to the hospital, with a history by the mother that during the interval, convalescence had been normal until two days ago, when the child began to vomit food and she noticed a staring look in the eyes. On admission the child had a temperature of 101°.

*Read before the New York Academy of Medicine, Section of Otology, March 13, 1931.
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Pulse, 120; definite rigidity of the neck, bulging of the anterior fontanelle, no abnormal ocular findings and the reflexes were normal at this time. There was a profuse mucopurulent discharge from the ear canal.

Nov. 4: Hemoglobin, 63 per cent; R. B. C., 3,600,000; W. B. C., 5,500; polys., 54 per cent; small lymphs., 44 per cent; trans., 1 per cent. Spinal fluid: It was very difficult to get any fluid, there being an apparent blocking above. The fluid obtained was too bloody for examination. Wassermann negative, Cultivation negative at 24 hours. Urine negative.

*Consultation:* The case was seen by Dr. Craig and Dr. Walter L. Carr on Nov. 5, the day before an exploratory operation was done. Dr. Carr reported the eye grounds negative, reflexes quick, but no abnormality. Rigid neck, but not always the same. Tentative diagnosis: *1.* Localized meningitis. *2.* Tuberculous meningitis.

Nov. 6: The child continued to vomit food. Exploration of the right mastoid was done.

*Findings:* Granulations filled the middle ear. There was a moderate amount of thick mucopurulent, pulsating discharge seen in the ear canal. The malleus and incus were removed to facilitate drainage. The squama, tegmen and the bone in front of and behind the sinus were removed over a large area, revealing the dura of the middle fossa moderately injected, but no granulations present. The pressure of the dura was considerably increased. The cerebellar dura and sinus appeared normal. The bone of the tegmen was softened. There was considerable bleeding from the superior petrosal sinus, so the wound was packed with iodoform gauze and left wide open. Following the exploratory operation the temperature went up to 102.3° but came down the following day to 100°. The child seemed to be better, he did not vomit and was taking his nourishment well. Temperature remained around 101°; pulse, 135-140.

Nov. 7: Hemoglobin, 61 per cent; R. B. C., 4,000,000; W. B. C., 11,000; polys., 75 per cent. Daily examination of the blood was made until Nov. 12, when the hemoglobin was 49 per cent; R. B. C., 3,600,000; W. B. C., 12,000; polys., 55 per cent.

Nov. 8: Spinal fluid was under no pressure and on examination revealed straw-colored (blood present) fluid; Fehling not reduced, 90 monolocular type cells to c.m.m.

*Bedside Notes,* Nov. 10: Wound dressed. Healthy granulations. Moderate discharge in the ear canal. Fontanalle unchanged. Still under considerable pressure. There seems to be a weakness of the left external rectus. Opisthotonus much less. Babinski: right, negative; left, positive. Quite drowsy today. Patient taking food well. No

vomiting since the operation. Slight discharge from the opposite ear, slight fullness of drum, left myringotomy.

Nov. 11: The child had several generalized convulsions this morning, unable to retain any food, condition seems worse. Pulse rapid, thready and irregular. Lumbar puncture was done and for the first time fluid was found under increased pressure. Child seemed considerably relieved by the puncture and the convulsions ceased. The spinal fluid at this time did not reduce Fehling, contained 50 mononuclear type cells per c.m.m. and no bacteria were seen in a centrifuged specimen. Cultivation negative. Eye examination, under atropin: Paresis of left external rectus persists. Fundus examination negative, except for a moderate anemia of both discs, which is due to the secondary anemia of the child.

*Consultation Notes,* Nov. 15, Dr. Joyner: It is quite evident this child has considerable meningeal irritation, but I do not believe there is an abscess localized at the present time. Advise daily lumbar puncture of as much fluid as possible, also daily cell count.

Dr. Craig suggested a supravital blood count for tuberculosis. This was negative. The supravital test for tuberculosis has been developed by the Rockefeller Institute and we are indebted to them for their co-operation.

The temperature remained around 101°,with remissions to 99°, with pulse becoming weaker, until Nov. 15, 1930, when the temperature went up to 104° and the patient died.

*Postmortem Findings:* The exposed dura, which was covered with granulations, was incised widely. The dura was considerably thickened. The brain tissue in the temporosphenoidal area and posterior fossa was explored without any evidence of an abscess.

A needle was then injected in the right lateral ventricle and about 50 c.c. of clear cerebrospinal fluid removed. This fluid was clear, in contrast to the amber fluid removed from the lumbar region.

Mr. Burchell reported the fluid from the lateral ventricle to contain streptococci, which was the same organism reported in the aural smear made six weeks before.

It was not possible to get consent for a complete autopsy.

In searching the literature, I do not find any cases of acute internal hydrocephalus complicating acute mastoiditis.

*Impressions:* I believe the streptococcus infection from the mastoid was transmitted through the blood stream to the choroid plexus of the ventricles, causing a plastic inflammation of the foramen of Magendie and Luschka. Possibly there was an ependymitis of the iter between the third and fourth ventricles, causing a blocking of the iter, thus producing the internal hydrocephalus.

30 W. 59th Street.

# MASTOIDITIS IN THE PRESENCE OF UNSUSPECTED DIABETES. REPORT OF A CASE.*

DR. GEORGE W. MACKENZIE, Philadelphia.

There are at least four different factors which can act as inhibiting influences to recovery from acute infections:

*1.* Improper diet, in other words, one that is deficient in vitamins, not the least important of which is fat soluble Vitamin A.

*2.* Insufficient ventilation, *a.* of the room in which the patient is confined; *b.* of the surface of the body because of unclean skin; *c.* of the infected area itself from retention of secretion.

*3.* Lack of sunshine, that most important vitamin restorative, fixing agent and vitality generator, the effect of which is determinable by the total white cell count.

*4.* The presence of one of the dyscrasias.

As the writer understands it, a dyscrasia is that depreciation of bodily resistance induced by the presence of a chronic ailment which makes the individual more vulnerable to an intercurrent acute infection and more liable to succumb to it than another individual who is not so afflicted.

Any of the chronic diseases can act as a dyscrasia so long as it remains latent in form. That individual with a dyscrasia which can cause an intercurrent pneumonia to take on a malignant form from the start and death within 72 hours may, because of the very latency of his chronic ailment, be entirely unaware of the danger threatening him. He may feel so fit as to be even boastful of his health.

Since it is the latent form of chronic diseases which acts as dyscrasias, the question naturally arises, what about the more manifest forms of these same chronic diseases? The answer is: When a previously latent chronic disease assumes the manifest form, it does so by reason of an acute exacerbation or flare up. The patient's condition becomes decidedly worse. He has already entered the stage of progression from which few ever recover.

Those dyscrasias with which the writer has had to contend in the treatment of what would otherwise have been nothing more than the ordinary run of cases were tuberculosis, syphilis, diabetes and

*Read before the Philadelphia Laryngological Society, March 3, 1931.

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focal infection. He has come to recognize the importance of these dyscrasias as distinctly inhibitive factors to recovery from acute infections about the ear and sinuses.

From time to time he has reported cases in which these dyscrasias played an important rôle but none has proved more instructive than the one to be reported this evening.

*The Case**: Patient, male, age 41 years, general surgeon somewhere in Pennsylvania, was first seen by the writer on May 16, 1930; called in for a consultation with the previous otolaryngologist and to operate upon the mastoid in the event that an operation was found indicated.

The following history was obtained: Three weeks ago the patient began with a tonsillitis, more severe on the right side. One week later the right ear started to pain. At that time a local specialist was called in, and he incised the drumhead. This was followed by some relief of symptoms. The temperature continued above normal in spite of the paracentesis. Narcotics were administered freely upon the beginning of his ear trouble. The general appearance of the patient was bad, in that he was quite pale (toxic, according to the resident physician). He wore an expression of distress and anxiety. His outstanding complaint was that of heavy pressure and pain in the right ear and mastoid region. There was thickening of the skin and soft parts over the right mastoid; besides, there was tenderness. There was profuse purulent, yellow, nonoffensive discharge from the right ear. Cleansing of the ear permitted a more careful inspection when there was revealed some sagging of the posterior superior wall of the osseous canal and a pulsating secretion from a perforation in the posterior half of the drumhead. Further landmarks were not discernible because of the narrowing of the canal and general swelling of the drumhead.

The X-ray diagnosis made just previous to the writer's visit was that of mastoiditis with beginning softening of the bony trabeculae.

There was something about the patient's general appearance, together with his overweight, that prompted an inquiry as to the amount of blood sugar the patient was carrying. The hospital records revealed the presence of but 111 m.g. of sugar per 100 c.c. of blood on the morning of the same day; besides, there was an absence of any sugar in the urine, neither was there any albumin. Other laboratory reports were negative.

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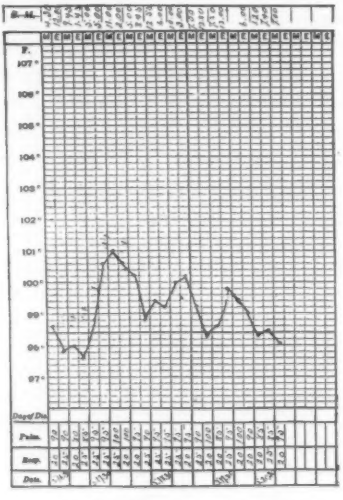
*The records in this case are so voluminous that it was thought best to abridge the report appreciably, recounting that much only which is necessary for our present purpose as covered by the title.

The brother of the patient, an X-ray specialist, practicing in Philadelphia, confirmed the opinion of the local X-ray man regarding the presence of a surgical mastoid.

The patient was prepared for operation at the completion of the examination. The operation was performed under gas-oxygen anesthesia, administered by Dr. Everett Tyler, of Philadelphia. Practically all cells were found involved with pus and granulations. Furthermore, softened bone was found, especially behind the external canal. The involved cells reached far into the zygomatic process, also in a median and posterior direction. The exposure of the middle and posterior skull fossae were made necessary by reason of the

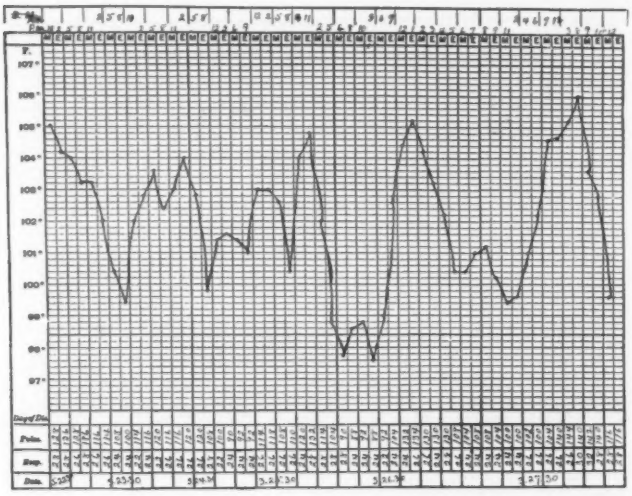


extensive destruction. The brother of the patient, who was present at the operation, remarked that he had never before seen such a wide distribution of mastoid cells. The appearance of the wound cavity suggested the presence of diabetes and for this reason further tests for diabetes were ordered.

The patient was again seen by the writer (May 18) 36 hours after the operation when the temperature was found to be 100.2° axillary, respiration, 20, and pulse, 90. Facial paralysis had developed on the right side. The outer dressings were changed. The lower silkworm suture was removed. The edge of the wound was noticeably pale. May 19: The facial paralysis was less marked.

Wound was redressed. May 20: The facial paralysis is the same. There are no complaints. Temperature, 99.8° axillary.

The next heard from the case was through a telephone call made by the brother, who asked if it would not be just as well to have the patient brought to Philadelphia for further redressing. This was agreed to and the next redressing was made on May 21. It was observed that the wound still showed the appearance of what the writer has come to look upon as characteristic of diabetes. In other words, the wound was pale and lifeless looking and the dressings came out of the wound without one drop of blood. At the time of this redressing the temperature by mouth was 98.4°. The same after-



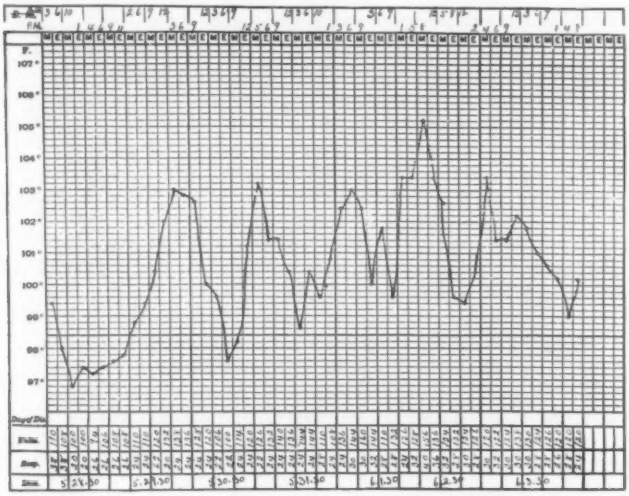
noon, at 3 o'clock, the brother phoned that the patient's temperature had gone to 101.4°, pulse, 120; chills at intervals, no pain. He was visited at his brother's home at 5 p. m., when conditions were found to be about the same as they were at 3 p. m. He was seen again the following morning, May 22, when it was reported that the temperature had gone to 102.4° the night before, but this morning it had dropped to 101.4°, pulse, 120. An enema had been given earlier, after which he claimed to feel much better. The facial paralysis appeared to be somewhat improved. The wound cavity presented the same general lifeless appearance. At 11 a. m. it was reported by phone that the patient's temperature had suddenly gone to 104°. It was

then advised that he be sent to the hospital and a consultation with an internist was suggested. The family selected Dr. G. Morris Golden. After a thorough examination his recorded opinion reads: "The patient's condition is suggestive of septicemia."

The blood count of May 22 revealed the following: Hemoglobin, 80 per cent; red cells, 3,560,000; white cells, 10,600; polys., 87 per cent; lymphocytes, 11 per cent; transitional, 2 per cent.

Urinalysis: Specific gravity, 1.022; albumin, ++; sugar, negative; acetone, negative; microscopic, epithelium, mucus threads, few pus cells.

Blood sugar test and Wassermann, blood culture and culture of pus from wound were also requested, but not made.



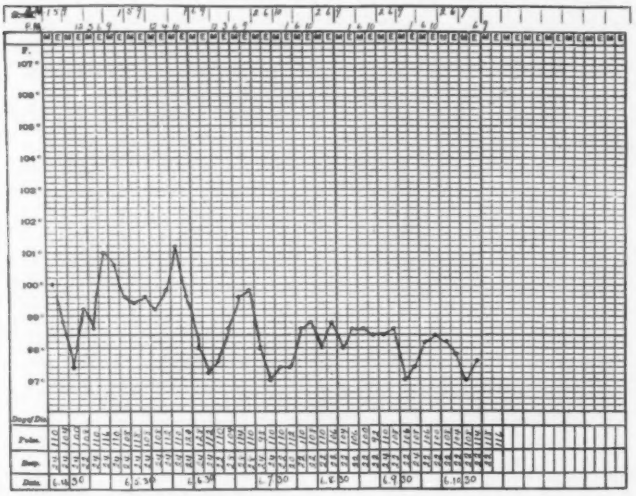
It was agreed that the internist should look after the prescribing of drugs and the regulation of the diet, particularly after the diagnosis of diabetes had been confirmed by the blood sugar test, which test was postponed for 11 days after the request had been made by both Dr. Golden and the writer.

May 22, a citrated blood transfusion, 250 c.c., was given. On the twenty-third there was a second citrated blood transfusion, 300 c.c. May 24, the patient received a third citrated blood transfusion, 250 c.c.

May 22, the highest recorded temperature was 105° at noon. May 23, highest temperature was 103° at noon. The lowest tem-

perature was 99.4°. May 24, the highest temperature was 104° and the lowest 99.8°. May 25, the highest temperature was 104.8° and the lowest 97.6°. May 26, the highest temperature was 105.2°, the lowest was 99.4°. May 27, the highest temperature was 106°, the lowest 99.6°. The rise and fall of temperature from that time on was sharp and characteristic of pyemia. The temperature reached about the normal point and remained there on June 7. See temperature chart. The highest recorded pulse was on May 31, when it reached 160, and the lowest pulse rate while in the hospital was 92; see chart.

Blood cultures were made daily and were negative until May 26, when the report came back from the laboratory, "full growth long-



chain streptococcus," which matched up with the culture of the wound secretion made on the same day. Although there was enough evidence to ligate the jugular and open the sinus before May 26, the indications were not convincing enough for the family and the internist until after the finding of the positive blood culture.

Intravenous injections of Pregl's iodin solution was given on two occasions. There was an improvement in the temperature after each injection of 10 c.c. of the iodin solution, the effects of which were rather confirmatory of a blood stream infection in the opinion of the writer. Following the ligation of the jugular the sinus was laid bare

from the jugular bulb to the torcular. When the sinus was opened the clot was found to extend the whole length of the exposed sinus. It was only after the removal of the most posterior part that free bleeding began. During the operation the patient's pulse rose to 180, when his condition was reported critical by the anesthetist. Immediately following the operation the patient was given a 300 c.c. transfusion of citrated blood.

Improvement began almost immediately following the operation. However, the progress was rather slow. Just before the operation upon the sinus the patient developed a pyemic abscess in the left shoulder, which was the only abscess to manifest itself. The temperature reached normal on June 7, after which there occurred no further rises.

Following the second operation, on May 27, the wound continued to look unhealthy, with no signs of normal granulations. The request for a blood sugar test was repeated and it was not until June 2 that it was acceded to. The report came back: blood sugar, 250 m.g. With a rigorous diet and without insulin, the blood sugar came down to 173 on June 5, and to 133 on June 9. By June 22 it had diminished to 95 m.g.

Summarized briefly, the outstanding points in this case are:

*1.* An apparently healthy and robust looking individual was stricken with tonsillitis, followed by a middle ear suppuration and mastoiditis. The infecting micro-organism was pronounced at the first hospital as the hemolytic streptococcus.

*2.* At the time of the first operation, because of the apparent lifelessness of the involved mastoid, diabetes was suspected but the suspicion was not confirmed by urinalysis or a blood sugar test made that same morning.

*3.* General appearance of the wound cavity, two days after the operation and again five days after the operation, presented the same appearance as that previously observed in wounds of other diabetic patients.

*4.* The rise of temperature on the afternoon of the fifth day and for two or three days afterward, with accompanying chills, was so characteristic of thrombophlebitis that the writer was prompted to operate. However, he waited long enough to receive a positive blood culture in order to satisfy the family.

*5.* Although the diabetes was more than suspected from the start, the lack of co-operation from the pathologist was responsible for the unnecessary delay in treating the case from that angle. Aside from the two operations, the treatment consisted in the free use of quinin

internally, blood transfusions, two injections, one on the twenty-seventh and one on the twenty-eighth, of scarlet fever streptococcus antitoxin, 15 c.c., 5,000 units intramuscularly, recommended by Dr. John A. Kolmer, who was in consultation on the case. Pregl's solution was injected intravenously, 10 c.c. on May 24, and 10 c.c. on May 26.

6. The wound presented the same lifeless, pale, dry appearance from the time he was first seen until June 2. Because of the high blood sugar (250 m.g.) the internist modified the diet to meet this exigency, from which time the mastoid wound cavity began to show an entirely different appearance. The excellent work of Dr. Golden in regulating the diet was responsible for bringing about the marked change in the patient's general condition, besides the improvement of the wound reaction.

In closing, the writer wishes to call attention to the importance of diabetes as a complicating dyscrasia to be reckoned with in all acute pyogenic infections calling for surgical intervention. It is a complication that is not difficult to recognize clinically; the anemia, scanty secretion and lifelessness of the wound being quite characteristic.

In any case in which there is a lack of red granulations after ten days or two weeks following an operation on the mastoid and signs of the destructive process extending beyond the original limits of involvement, one is justified in thinking of diabetes.

There are more cases of this kind than the average otologist is inclined to believe. Vigilance is the price of success. When one has a "hunch," so to speak, that there might be a complication, it is not well to wait until all of the characteristic symptoms have manifested themselves, for if we do it is generally too late.

It is well, too, to bear in mind that negative findings prove nothing. If they do, then the patient whose case was just reported did not have diabetes, since there was no sugar in the urine and no excess in his blood the day he was first operated.

The differentiation of the dyscrasias as complicating factors in mastoiditis is too big a subject to even touch upon at this time.

1724 Spruce Street.

# ACUTE MASTOIDITIS ASSOCIATED WITH HEMATURIA.*

DR. MATTHEW S. ERSNER and DR. EDWARD K. MITCHELL,
Philadelphia.

After analyzing 81 mastoids performed at Temple University Hospital in the past year, it was found that three cases presented hematuria at time of operation. In perusing the literature we were unable to find any such observations. Our findings, therefore, prompted us to report these cases for your consideration.

Attention has been drawn to the upper respiratory tract as a source of focal infection but the ear has been somewhat neglected. As far back as 1898 Hartman called attention to the fact that infants with ear infection often suffered with diarrhea and that these symptoms improved with paracentesis. It remained, however, for Maurice Renauld, of Paris, in 1921, who seems to have been the first, to observe that this infection may involve the mastoid antrum. In 1922 the author (Ersner) reported a group of cases presenting atypical mastoiditis associated with gastrointestinal disturbances complicating the mastoiditis (published in the *Atlantic Monthly*, 1922). In 1925 Lyman and Alden, of St. Louis, presented a series of cases of infants with diarrhea, vomiting and loss in weight, due to a streptococcic hemolytic infection of the mastoid.

To date little or no attention has been called to the fact that acute mastoiditis, particularly of the so-called hemorrhagic type, may act as the causative factor in the production of hematuria.

In the course of analysis of 81 mastoids at Temple University Hospital on our service it was discovered that three cases presented hematuria associated with acute mastoid infection. Two of the cases had microscopic hematuria and one presented a marked macroscopic hematuria. In these three cases the diagnosis of hemorrhagic mastoiditis was made at the time of operation and the streptococcic hemolyticus was grown on culture from the mastoids. Unfortunately, cultures of the urine were not done. The striking feature was the disappearance of the hematuria following drainage of the mastoid. The following are the histories under discussion:

L. F. male, white, age 7 years, admitted to the Temple University Hospital on May 17, 1930, complaining of pain and discharge from

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the right ear. He was well until three weeks prior to admission, when he was seized with sudden pain in the right ear. A myringotomy was performed, followed by discharge of creamy, white pus. Drainage continued for one week, then ceased. The eardrum was reincised, followed again by a suppuration. Three days prior to admission he began complaining of severe headache in the right temporal region.

Examination revealed slight auricular displacement, loss of postauricular folds and tenderness over the tip of the mastoid. The eardrum presented a central perforation, with sagging of the posterior superior canal wall. The left ear was negative.

Temperature, 100.4°; pulse, 100; respiration, 20.

Heart and lungs negative.

Urinalysis showed a specific gravity of 1010, acid, faint trace of albumin, negative sugar, no white blood cells, but many red cells.

Blood count revealed: White blood cells, 14,400; polymorphonuclear, 84 per cent; small lymphocytes, 12 per cent; large lymphocytes, 2 per cent; eosinophiles, 2 per cent.

A simple right mastoidectomy was performed on day of admission to the hospital, under ethylene anesthesia. The findings at the time of operation were as follows:

*1.* Thickened cortex. *2.* Pus and granulation tissue throughout the mastoid cells. *3.* Necrosis, mostly marked at tip of mastoid. *4.* Hemorrhagic type.

Culture from mastoid revealed the streptococcus hemolyticus.

Two days later the temperature dropped to 99° F.

Urinalysis at this time showed a specific gravity of 1015, alkaline, negative albumin, negative sugar, few epithelial cells, negative white blood cells, no red blood cells. Patient was discharged on the seventh day in good condition; mastoid wound healed, no discharge from the right ear, urinalysis negative.

*Case 2:* A. P., female, age 15 years, admitted to Temple University Hospital on Jan. 3, 1930, complaining of pain in back of right ear, with a suppurative discharge from the same ear for six days prior to admission. Her temperature on admission was 101.2°. There was postauricular edema over the right mastoid, with tenderness over the antrum and tip. The white blood count was 15,800; polymorphonuclears, 79 per cent; small lymphocytes, 12 per cent; large lymphocytes, 8 per cent; eosinophiles, 1 per cent.

Urinalysis showed an amber-colored urine acid reaction, albumin trace, sugar negative, few white blood cells and many red blood cells. (In this case the urine was carefully collected so as to avoid bloody contamination from the genital organs.)

A simple mastoidectomy was performed under nitrous-oxid-oxygen ether anesthesia. The operative findings were as follows: *1.* Thickened cortex (hemorrhagic in character). *2.* Scattered areas of empyema of the mastoid cells. *3.* Antrum filled with pus and granulation tissue. *4.* Lateral sinus exposed for about an eighth of an inch.

Three days after operation the urinary findings were as follows: Specific gravity 1018, acid, faint trace of albumin, negative sugar, occasional white cells and no red blood cells. Repeated urinary examinations failed to show the presence of hematuria.

*Case 3:* P. T., age 67 years, female, admitted to Temple University Hospital on May 10, 1930, complaining of discharge from both ears and pain along the left side of the neck. The condition began two months prior to admission but had become worse in past two weeks. Patient was a diabetic for the past ten years.

Examination revealed an elderly woman in poor state of nutrition, completely blind, temperature 100°, both mastoids were edematous and tender. Large fluctuating mass felt over and beneath tip of left mastoid. A diagnosis of acute bilateral mastoiditis with a left Bezold's abscess was made.

Blood count showed: Leukocytes, 11,900; polymorphonuclears, 85 per cent; small lymphocytes, 8 per cent; large lymphocytes, 3 per cent, and eosinophiles, 4 per cent.

Urinalysis revealed specific gravity 1020, acid, faint trace of albumin, sugar, 5 per cent; few hyaline casts, occasional white blood cells and an abundance of red blood cells.

After having attempted to counteract the acidosis present by means of insulin and glucose under the direction of the medical department, a bilateral mastoidectomy was performed under ethylene and oxygen anesthesia. Operation revealed pus and granulation tissue in both mastoids, presence of sequestra and a left Bezold's abscess. The mastoids were of the hemorrhagic type.

## SUMMARY.

In dealing with hematuria we must consider its various causes. Not only does the kidney pathology, *per se*, produce hematuria, but systemic conditions as hemophilia, purpura hemorrhagica, leukemia, Hodgkin's disease, scurvy and such irritant drugs as cantharids, turpentin and prolonged use of urotropin. By means of the history and clinical findings we are able to eliminate the above as the etiology.

Otogenic infections caused by the streptococcus hemolyticus may produce many complications, *i. e.*, sinus thrombosis, endocarditis, nephritis, *et cetera*. In the three aforementioned cases, hematuria

resulted. It is problematical whether the hematuria resulted from the mastoiditis or whether it was focal or embolic in origin; on the other hand, it may have been the result of a direct attack by the toxins on the kidneys. If I may digress I should like to reiterate the interesting features of the cases on discussion. *1.* Hemorrhagic type of mastoids. *2.* The streptococcus hemolyticus was isolated from the mastoid wounds. *3.* The presence of the eosinophil in the blood picture; and last, but not the least, *4.* Hematuria.

From our statistics we learned that approximately 50 to 60 per cent of all operative mastoids were due to some form of streptococcus hemolyticus. Many observers have called our attention to the fact that certain streptococcic hemolytic strains are prone to produce complications. From a previous research, we learned that all positive cultures obtained in sinus thrombosis were found to be due to the streptococcus hemolyticus. It is therefore possible that certain streptococcic hemolytic strains found in mastoiditis and sinus thrombosis bears a close relationship to the scarlatinal organism. If the latter be true, many complications can be explained. In these three particular cases hematuria resulted.

The presence of the eosinophil is of utmost significance. It is a well established fact that the eosinophil belongs to the leukohematopoietic system. The rôle that the eosinophil plays is not entirely understood. It is normally present in the blood, disappears during acute infection, reappears when the infection subsides and is constantly present in allergic phenomena. The allergic disturbances may be exogenous or endogenous. In the hemorrhagic mastoid caused by the streptococcus hemolyticus, we have endogenous lytic elements produced by the organisms affecting the red blood system and result in a hemolysis. In the course of the mastoid destruction lytic elements are produced and liberated in the mastoid area. Therefore, the presence of the eosinophil in the above enumerated cases was a forewarning of lytic phenomena. The hematuria here may be just another lytic element added.

Since there was an elapse of time from the onset of infection to the time of operation, the streptococci no doubt spread its toxins to other organs, and in these particular cases the urinary tract was involved. There is no doubt that the hematuria was the result of the infection, as it completely disappeared following operation. This finding was substantiated by repeated postoperative urinary examinations.

In speaking of the influence of focal infection, we are dealing largely with theoretical considerations and not with definite, demonstrable facts. It is only by analyzing and deducting that we arrive

at conclusions. However, hematuria concomitant with mastoiditis should be considered as one of the indications for mastoidectomy.

The history, physical findings and progress of these cases lead us to believe that the ear and the mastoid should be taken into consideration as a source of focal infection.

CONCLUSIONS.

1. Acute mastoiditis associated with hematuria must be looked upon with suspicion.

2. The hematuria was transient in these cases, disappearing after mastoidectomy.

3. The mastoids were of the hemorrhagic type, with recovery of the streptococcus hemolyticus on culture from the mastoid.

4. Emphasizing the mastoid as a focus of infection causing hematuria.

5. Persistent hematuria associated with mastoiditis should be regarded as another positive indication for mastoidectomy.

1915 Spruce Street.

710 W. Lehigh Avenue.

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## CARCINOMA OF THE LARYNX: REPORT OF AN UNUSUAL CASE.*

DR. LOUIS H. CLERF, Philadelphia.

If the larynx of every patient with hoarseness was examined and a biopsy made when indicated, a majority of the cases of laryngeal carcinoma could be correctly diagnosed in the early stages of the disease. The reason for this is apparent when one recalls that in a majority of cases the cancer is intrinsic and most commonly involves the vocal cords or subglottic tissues. Of the intrinsic cases, about 90 per cent either have hoarseness or some alteration of the voice as the first symptom.

Failure to make an early correct diagnosis may be due to one of many causes. The widespread cancer propaganda being carried out

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is doing much to educate the laity concerning the need of prompt investigation of hoarseness. Other factors are failure to examine the larynx before prescribing treatment, overemphasis of the cancer age and the occurrence of cancer in atypical locations.

For many years the age of the patient was stressed in cancer diagnosis; cancer was considered as being rare before the age of 40 years. It is a question whether age should be too seriously considered in the diagnosis of cancer. If a laryngeal lesion is suggestive of cancer it should be investigated from that standpoint, irrespective of the patient's age. MacKenty reported a case of laryngeal cancer occurring in a girl, age 20 years, and a man, age 29 years. Others have reported cases of cancer occurring during the early thirties.

Fig. 1. A mirror view of the larynx before direct laryngoscopy was done. A small part of the right side of the larynx could be seen. It was impossible to determine the point of attachment of the growth. Its upper surface was undergoing necrotic changes, so that its appearances were not characteristic of any definite clinical picture.

Although cancer commonly occurs in the anterior two-thirds of a vocal cord it may occur in any locality. Here one must be careful in expressing an opinion. Atypical cases should always be regarded with suspicion and a final conclusion should be promptly arrived at if possible.

The following case is unusual because of the age of the patient, the location of the lesion and the brief duration and character of the symptoms complained of.

*Case Report:* Female, age 21 years, a college student, was referred to me four weeks after she had noticed some distress on swallowing and a sensation of a lump in her throat. She was busy with final examinations, so did not consult a physician until two weeks after the onset of the symptoms.

When admitted to the Bronchoscopic Clinic there was moderate disturbance in swallowing, frequent clearing of the throat and expectoration of mucus. The distress on swallowing was due to interference with the protective function of the larynx and not to obstruction of the food passages. The breath was fetid. There was no hoarseness nor dyspnea; the voice had a distinct muffled sound. By mirror laryngoscopy there was found a large, irregularly globular, sloughing mass, which obscured a view of the larynx and seemed to completely fill its upper orifice. The epiglottis and a portion of the right arytenoid were visible (see Fig. 1). Nothing abnormal was discovered by general examination and systemic tests. A direct laryngoscopy was performed. The growth was found to spring from the apex of the left arytenoid and the left aryepiglottic fold. Curiously, the motility of the arytenoid was good. A considerable portion of the growth was removed to relieve the swallowing disturbances and for histologic examination. Following this, the voice was normal and swallowing was undisturbed.

Histologic study, using the rapid paraffin method, was completed within 24 hours, and Dr. B. L. Crawford reported the tumor as a squamous cell carcinoma, anaplastic in type.

The type of tumor rendered the prognosis unfavorable. The question of laryngectomy was discussed with the family and patient and this plan of treatment was elected. The growth was found to be limited to the arytenoid and corresponding aryepiglottic fold without extension to the base of the epiglottis. No metastasis was apparent.

*Comment:* The age of the patient should be of statistical interest; it should not be too seriously considered in a case where cancer is suspected. Atypical appearances and unusual locations of lesions often lead to errors in diagnosis and call for a complete and thorough study before arriving at any final conclusions.

128 S. Tenth Street.

# EXPOSURE OF SIMULATED DEAFNESS BY BONE CONDUCTION TESTS.*

DR. B. M. BECKER, Brooklyn.

Up to the present time all attempts for the detection of malingering were made by means of air conduction tests. It is true that a vibrating tuning fork placed on the midskull (Weber) is made use of by some otologists in testing for simulated deafness, but its results are not instrumental in determining the status of the auditory function in the individual thus tested. The best that is claimed for the method is that one gains certain useful impressions as a result of its use.

In speaking of the tests employed in unmasking simulated deafness, Kerrison states (Diseases of the Ear, 4th Edition, pp. 94-95, "Weber's Test"): I now apply a vibrating tuning fork of 256 double vibrations to the vertex of the skull, and if he refers the sound to his supposedly deaf ear, I am favorably impressed as to his honesty. If he refers it to the sound ear, I become skeptical, and proceed to the next test."

The limited value of each separate bone conduction test, whether used for general functional testing or for the purpose of uncovering pretended deafness, was due, to a great extent, to the fact that under no condition could one ear be completely ruled out while testing the other, as is done in air conduction tests by means of the Barany noise apparatus. As a consequence of this an element of uncertainty always injected itself in the interpretations of the results obtained from them.

In the method to be described presently, the Weber test is employed, not as a means of obtaining impressions as to the probable honesty or dishonesty of the patient, but rather as a means of determining the nature of his deafness (perceptive or conductive), taking the statements of the patient for granted until by further tests he is proved to be either a sufferer or a malingerer.

*Principle:* When a current of air is directed steadily and forcefully against either tympanic membrane, no extraneous sound can reach the cochlea of that ear either through air or bone conduction. The ear is temporarily rendered deaf to all sound save that which is produced by the air current.

*From the Departments of Otolaryngology, the Long Island College and the United Israel-Zion Hospitals.

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*Procedure:* One free end of a soft rubber tube is put close to the ear canal of the patient, and the other free end is placed between the lips if the examiner, an assistant, or the patient; and by forced expiration, a steady current of air is directed against the drumhead of the respective ear. (The same results will be obtained by the use of a compressed air tank or a spraying outfit which can deliver a volume of air equal to that of the lungs.) A strongly vibrating tuning fork, 256 or 128 double vibrations, is set on the vertex or on either mastoid antrum, and the usual questions are put to the patient. If the examiner and patient cannot freely communicate during the examination because of the tube in the mouth of either one, the patient is instructed beforehand to raise a finger as an indication of his hearing the sound, and to point it to the right or left ear, depending upon the localization of the sound to one side or the other. For the sake of brevity and ease of recording, the examiner may mark the results of the examination by means of symbols and abbreviations. Thus: When the patient indicates that he does not hear the sound, a simple minus sign (—) is sufficient; when he does hear it, a plus symbol (+); when he hears it but cannot localize it, an indeterminate symbol (±); when the sound is heard to the right (+R), and when to the left (+L).

*Example:* A patient presents himself for examination, complaining of total loss of hearing in the right ear. On examination, no visible pathology or abnormality is discoverable in the meatus or drum, and to all the hearing tests by air conduction he returns a negative reply. A vibrating fork placed on the skull gives a localization to the left ear as a result. A similar result is obtained with the fork on the right mastoid. Here we are confronted with a patient who is suffering either from a grave lesion of the right perceptive mechanism, or is simulating that condition. At this point, the method as outlined above is made use of. The tube is placed in his right ear and a current of air is directed against his drum, while a 256 or 128 vibrating fork is pressed against his right mastoid. The true sufferer from perceptive deafness will indicate that he hears the sound but he cannot determine on what side, or in some cases he will localize it to the left. The malingerer, although now he is on a par with the true sufferer, his right cochlea being rendered functionless by the air current against his drum, will either refer the sound to the left or deny hearing any sound, the reason for his denial being the fear of exposure. Not knowing that the fork on the right mastoid can be perceived in the opposite labyrinth he may regard an admission of hearing as an admission of guilt. With the tube still in the right ear the vibrating fork is now transferred to the left

mastoid, when both will unhesitatingly refer the sound to the left ear. Now the tube is placed in the left ear and the same procedure followed. When the fork is over either mastoid the true sufferer from a perceptive lesion will return a negative reply, for the reason that the right cochlea is functionless by virtue of the lesion, and the left is temporarily made functionless by the air currents so that he is rendered totally deaf binaurally. The malingerer, on the other hand, under the same condition of testing, will refer the fork sound, either when placed on the right or left mastoid, to the left ear. For, hearing the sound and not knowing that his left ear is made deaf, he will return a positive reply. In his answers to the last procedure lies the crux of the test, which spells his undoing.

For the sake of comparison and ready reference a tabulated chart is herewith appended representing four types of individuals subjected to bone conduction tests with their responses to them.

*A.* One with normal bilateral hearing.

*B.* One with a conductive lesion in right ear, left ear normal.

*C.* One with perceptive lesion in right ear, left normal.

*D.* One with a simulated perceptive lesion in right ear (malingerer).

| *Fork on vertex:* | *Fork on right mastoid:* | *Fork on left mastoid:* |
|---|---|---|
| (A) ± | (A) +R | (A) +L |
| (B) +R | (B) +R | (B) +L or +R |
| (C) +L | (C) ± or +L | (C) +L |
| (D) +L | (D) — or +L | (D) +L |

With the tube in right ear and air current directed against right drum:

| *Fork on right mastoid:* | *Fork on left mastoid:* |
|---|---|
| (A) ± | (A) +L |
| (B) ± | (B) +L |
| (C) ± | (C) +L |
| (D) —or + L | (D) +L |

With the tube in left ear and air current directed against left drum:

| *Fork on right mastoid:* | *Fork on left mastoid:* |
|---|---|
| (A) +R | (A) ± |
| (B) +R | (B) +R |
| (C) — | (C) — |
| (D) +L | (D) +L |

By comparing the answers of the patient under examination with the schematic tabulation as outlined above one can at a glance detect any discrepancies found and draw his conclusions therefrom.

4802 18th Avenue.

# LYMPH HEMANGIOMA OF THE NOSE, PALATE AND TONGUE.*

Dr. Myron A. Zacks, Philadelphia.

Neoplasms of this type existing separately are not infrequent and may occur anywhere in the oropharynx. In combination, however, the case herein presented is not frequently observed, particularly from the standpoint of the otolaryngologist, and therefore merits report.

Such tumors are composed of dilated blood lymph vessels and spaces lined by endothelium. Strictly speaking, many of these are not genuine tumors but consist merely of localized dilatations and elongations of pre-existing vessels. Lymphangiomas consist of lymphatic dilatations as well as new growth of vessels. While actual enlargement and varicosity of existing lymph vessels may occur, it is quite exceptional to find it in the form of definitely circumscribed growths. Hemangiomas are observed chiefly in two varieties: the teleangiectatic, in which the blood vessels are merely distended, and the cavernous, in which the blood spaces are enlarged.

From a structural aspect, teleangiectatic angiomas are simply dilated capillaries lined with endothelium, reinforced by an outer fibrous coat. Most commonly, however, the vessels are considerably thickened and held together by a recticular connective tissue. The cavernous angioma presents large spaces lined with endothelial cells, between which spaces are found parallel fibres of connective tissue which forms the frame work of the tumor. The enlargements are usually congenital, essentially benign, and may remain throughout life without enlarging. However, occasionally after a few years, they may begin to increase in size and invade tissues quite some distance from the original site. Butlin, in mentioning angiomas of the tongue, reports a case of congenital angioma of the cavernous type which had grown to such size that it required excision of the whole anterior two-thirds of that organ.

While it is true that growths of this character are considered benign from the pathologist's point of view, it is well to bear in mind that secondary changes may occur in the connective tissue of the vessels of an angioma as in the plexiform angiosarcomata in

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which the blood vessels are surrounded by ensheathing sarcoma cells. Troublesome hemorrhage may follow slight irritation. They are also subect to inflammatory and necrotic changes which may cause general septic infection and in the tongue, acute glossitis and even edema of the glottis.

*Report of Case:* On Dec. 10, 1930, the patient, H. B., male, age 5½ years, was admitted to the Mount Sinai Hospital under the otolaryngological service of Dr. David N. Husik. Two days prior to admission, patient consulted the writer with the complaint of enlargement of the nose, and ulceration on the roof of the mouth. Present attack began three days prior to admission to the hospital, with pains in the stomach, nausea and vomiting, swelling and redness of the nose, soreness of the roof of the mouth, watery nasal discharge, feeling of malaise, severe headache and fever. Patient was put to bed, given a cathartic, and was considerably improved the following morning. Parents state that child was born with a peculiarly shaped nose. Patient has been a thumb-sucker since birth and it is his father's opinion that the ulceration on the roof of the mouth may be due to this habit. About the same time, one year ago, patient contracted a rather severe upper respiratory infection and developed a red nose with a large blister on the bulbous portion, which ruptured in a few days and discharged its watery content. A scab persisted on the nose for some time following this. During this attack the nose was very sore and considerably swollen. The roof of the mouth was not observed at that time. The family physician was consulted and suspected frost-bitten nose. All the symptoms subsided in the course of the next few weeks, when it was noted that the nose was smaller than ever. Parents state that the child occasionally traumatizes the nose and which is followed by immediate swelling. In the interval since the attack one year ago, the nose returned to almost normal shape and has remained so until the present attack. Except for the blister and scab noted in the previous attack, both attacks were more or less identical.

*Past Medical History:* Measles, mumps, 1930; cervical adenitis, T. and A., four-five months ago. About a year ago the father noticed a slight attack of hives involving the neck and ears. No other similar attack. No history of asthma or eczema. No abnormal gastrointestinal symptoms.

*Family History:* Essentially negative.

*Physical Examination:* Chest, abdomen and extremities negative for significant abnormalities.

*Nose:* Unusually large for an individual of this age because of a large bulbous tip which is somewhat darker in color than the rest

of the organ. On palpation it is painless to deep pressure, definite vascular irregularities are absent, and is cystic to touch. The inflammation seems definitely localized to the bulbous tip.

*Mouth:* When the lower lip is pulled forward the submucous veins are numerous and hugely distended with blood. Small, numerous, scattered teleangiectatic spots are seen on the buccal surface of both cheeks.

*Tongue:* Numerous teleangiectatic spots are present on the entire dorsum, particularly its anterior half. Beginning at about the middle third and extending posteriorly for practically the remaining portion of the organ, there is an irregularly elevated, verrucous growth, somewhat pale in color and studded with teleangiectatic spots. On palpation the growth is somewhat hard, irregular, and imparts a warty feel to the palpating finger. On the left side of the posterior portion of this lesion is found a fairly large area consisting of an hemangiomatous growth, port wine in color. The tongue is essentially otherwise normal in size.

*Palate:* At the junction of the hard and soft palate a superficially infected, ulceromembranous area is present, irregularly oval in outline, about 1 inch long and about a half-inch wide, surrounded by superficially placed blood vessels. This lesion is in direct contact with the growth on the tongue when this organ is elevated.

*Pharynx:* Evidence of a well performed tonsillectomy. Scattered teleangiectatic spots throughout pharyngeal mucosa.

*Laboratory Examination:* Urinalysis negative.

*Blood Examination:* Hemoglobin, 80 per cent; R. B. C., 4,670,000; W. B. C., 7,200; polys., 64 per cent; small mononuclears, 36 per cent.

*Blood Examination* (per 100 c.c. of blood): Urea nitrogen, 22 m.g.; sugar, 75 m.g.; plasma phosphorus, 3.92 m.g.; serum calcium, 12.5 m.g.

*Blood Wassermann:* Negative to Kolmer and Kahn tests.

*Blood Coagulation Time:* 4½ minutes.

*Treatment:* Mention is merely made of the various forms of treatment without regard, however, for their relative, respective, comparative merits; *e. g.*, X-ray, radium, electrocoagulation, fulgeration, actual cautery, carbon dioxid snow and excision. Surgical diathermy is preferred in vascular areas, such as the tongue, or the buccal mucosa. Since recurrence, particularly at the border of a previously cauterized area has been observed, removal must be radically wide.

Patient was discharged from the hospital after seven days and parents instructed to restrain the child's arms, particularly when asleep, so that the habit of thumb-sucking could be controlled. Under this regime for only one week, it was noted that the lesion of the palate had completely disappeared and in its place several pit marks were noted. Less attention was then given to the restraint of the arms, when, a month later, the lesion of the palate originally described, recurred fully developed.

When last seen, three days ago, the condition of the nose and tongue showed little, if any, change from its original condition; the palate showed moderate amount of recurrence.

COMMENT.

*1.* As a clinical entity the lesions described are apparently rarely encountered by the otolaryngologist. This is further borne out by the scarcity of literature dealing with such lesions.

*2.* Two distinct similar attacks within the past year. The blister mentioned in the first attack was probably of herpetic origin.

*3.* The skin is mentioned as the favorite seat, but subcutaneous tissues and sometimes the mucous membranes are involved.

*4.* Combined blood lymph tumor on the tongue, cavernous hemangioma of the nose, and teleangiectatic lesion of the palate.

*5.* Textbooks describe hemangiomas of the tongue as occurring on the anterior third. In our case, it occurred near the junction of the middle and posterior third, more particularly the posterior third.

*6.* Coexisting congenital and acquired similar tumors in the same individual. We feel justified in assuming that the tumor of the nose and tongue are of congenital origin, while the lesion of the palate has a traumatic background.

*7.* Bednar's ulcer is the title given to an ulceration found in the mouth, occasionally on the hard and soft palate, particularly in children, due to the irritation and traumatism of sucking, forcible cleaning with medicated applicators, use of hard rubber nipples in nursing children, etc.

*8.* While the lesion may be considered benign, it is subject to dangerous consequences and for this reason early complete eradication is advised.

1633 Spruce Street.

## International Digest of Current Otolaryngology.

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Heindl, Sr., of Vienna, in the May, 1930, Monatsschrift f. O., R. and L., writes on the recognition of tonsillar stones. He describes two cases and uses Zarniko's quotation as his concluding remark. This quotation is: "The appearance of an inflamed tonsil which contains a fistula should bring to mind the thought of neoplasm; then palpation with a sound and finger readily discloses the presence of a stone, which renders the differential diagnosis quite simple.

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It is interesting to note that in the September, 1930, Monatsschrift f. O., L. u .R., there are two articles on the uvula. This much neglected organ in our specialty has been receiving scant attention in recent years, and Marschik, of Vienna, presents a brief dissertation on the physiology of the uvula. Immediately following this article is one by Carl Bernfeld, of Jerusalem, who reports on anatomical and clinical investigation in acute and chronic nonspecific inflammations of the uvula. Both writers declare that their investigations have not as yet allowed a clearer understanding of the organ.

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Gustav Bruhl, of Berlin, in the September, 1930, Monatsschrift f. O., L. u. R., writes on rhinological endocranial complications. He observed six fatal cases from 1925 till 1929. The six cases were:

*1 and 2.* Meningitis following frontal sinus operation in acute frontal sinusitis.

*3.* Meningitis in chronic frontal sinusitis following trauma.

*4.* Meningitis with thrombosis of the longitudinal sinus following septum operation in acute frontal sinusitis.

*5.* Frontal lobe abscess following ethmoid operation.

*6.* Meningitis and subdural abscess in an untreated case of chronic frontal sinusitis.

He briefly describes each of his cases. In his discussion Bruhl strongly warns against a submucous resection in cases of fresh frontal sinus inflammation. External operation for these complications is the method of choice if any surgery is to be done.

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Hans Claus, of Berlin, in the September, 1930, Monatsschrift f. O., L. u. R., presents a paper on "Complications Following Tonsillitis." He has searched the literature for reports of pyemia, and presents a record of the cases to date with their results. He asks the prescribed question of why one patient should develop a localized peritonsillar abscess and another a pyemia. He is at a loss to explain how the large Vienna University Clinic was able to observe so few cases of pyemia without neck symptoms.

In the Berlin Clinic he was able to find 70 cases of pyemia following angina, in a period of about three and three-quarter years. He makes probable answer that the Berlin school is more interested in the subject and probably were looking for it, whereas the neck symptoms may have been missed in Vienna. He claims that there are probably still more cases which are missed and passed as pneumonia and sepsis of unknown origin. The aid of the internist, surgeon and specialist must be sought as any one alone probably may not see the other's part of the picture.

The problem to decide is whether or not the tonsils are to blame. If there is only a mild acute inflammation, and that is usually the case, then the history is of extreme importance.

What shall be indications for surgical interference? Long standing, subacute history of fever, with or without chills; pain on pressure over the cervical glands; pain over the sternocleidomastoid muscle; tenderness over the thyroid gland, are his main criteria. Differential diagnosis must be made between acute leukemia, agranulocytic angina, pyelitis, erysipelas and malaria. The above array of symptoms arising after scarlet fever angina should be operated on in the same fashion. The surgical treatment is external and search is made for the pocket of pus in the neck following treatment in the region of the sternocleidomastoid muscle from the processus mastoideus downwards. All diseased vessels are tied and resected where there is fear of emphysema due to air in the vessels.

In his conclusions he states that when a patient becomes septic following tonsillectomy or following an acute inflammation in the tonsils, it should be considered that there is a phlegmon arising which must be watched until it is possible to open it wherever it may point.

## BOOK REVIEWS.

**Atlas de Anatomia del Organo del Oido y de las Regiones Con el Vinculadas.** By Dr. Pedro Belou, Professor titular de Anatomia descriptiva de la Facultad de Ciencias Medicas de Buenos Aires. With 302 stereoscopic photographs, 159 photographs and 97 colored plates. Est. Grafico "Oceana," Buenos Aires, 1930.

This work is put up in a looseleaf folder of some 235 pages, each page being detachable for demonstration purposes. It has a foreword by Prof. Sebileau, who considers it an excellent work. The anatomy and morphology of the temporal bone is most exhaustively pictured and will be of great use to teachers of otology in demonstrating anatomical points of interest.

Many of the illustrations are stereoscopic and may be used for projection purposes. There are more than 300 such illustrations, many in color, which show the anatomy of the temporal bone. Despite the legends being in Spanish, many English-speaking anatomists and otologists will derive great benefit from the study of this atlas. M. F.

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**Hay-Fever, Hay-Asthma.** Its Causes, Diagnosis and Treatment. By William Lloyd, Fellow of the Royal College of Surgeons; Fellow of the Royal Society of Medicine; Late Surgeon-in-Charge of the Nose, Ear and Throat Department, Kensington General Hospital, St. Pancras and Great Northern Dispensary. Third Edition. London: Straker Brothers, Ltd., 194-200 Bishopsgate, London E.C.2.

This is the third edition of a monograph originally published in 1907. According to the author it has long been out of print. The cause of Hay-Fever is very clearly defined and liberal references to the early literature are made. In the treatment of Hay-Fever the author is decidedly against the use of vaccines or serums. The following is a quotation from his own preface of the third edition: "It will be seen, in connection with vaccines or serum treatments, that my results are practically negative; whereas with the line of treatment indicated—the result of an experience of over 30 years—in most cases cure, and in many cases relief, can be reasonably insured."

Hay-Fever usually produces sensitive areas in the nose; these areas may be recognized by increased coloration and slight raising-up of the mucosa. In his treatment the author cauterizes these areas. Oil of chamomile is used by nasal application, and the author is very enthusiastic about its use as a prophylactic if applied to the nasal passages some weeks before the Hay-Fever season. He uses the usual palliative nasal shrinking methods and routinely recommends a nutritious and easily digestible diet, and avoidance of over-exertion. Strong coffee on an empty stomach is recommended as of marked value during an attack of asthma.

Gross pathology in the nose and throat (sinus diseases, infected tonsils, deviated septum, etc.) is treated.

The author makes it a rule to wash out the antrum in all cases of Hay-Fever; the relief afforded is sometimes positively dramatic.

The reviewer of this book feels it is rather refreshing to find an experienced author who uses rational methods in his treatment of allergic conditions without going to the extremes of desensitization tests and inoculations. M. F.